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FOOD POISONING

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BACTERIAL FOOD POISONING

A CONCISE EXPOSITION OF THE ETIOLOGY, BACTERIOLOGY, PATHOLOGY, SYMPTOMATOLOGY, PROPHYLAXIS, AND TREATMENT OF

SO-CALLED PTOMAINE POISONING

BY

PROF. DR. A. DIEUDONNÉ

MUNICH

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NOTE BY THE AMERICAN AUTHOR

PUBLISHED less than a year ago, Professor Dieudonné's manual on "Bacterial Food Poisoning" has already become favorably known as one of the best presentations of the subject. In the present translation, the editor has incorporated descriptions of a number of additional outbreaks of food poisoning, elaborating upon the prophylaxis applicable to American conditions, and going more fully into the details of treatment. Paragraph headings have also been inserted, and the material slightly rearranged, so that the same natural sequence is followed in the different chapters. It is believed that these changes, together with the index which has been added, will facilitate reference and still further enhance the value of the book.

CHARLES BOLDUAN.

NEW YORK, January, 1909.

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INTRODUCTORY

ILLNESS due to the eating of infected foods is very frequent, in fact far more so than is generally believed, for usually it is only when a large number of such cases occur that the matter receives public notice. There is little doubt that many indefinite infections and disturbances of the intestinal tract, ordinarily termed errors in diet, are due to the ingestion of decayed or infected foods.

At one time most cases of poisoning through foodstuffs, especially meat poisonings, were thought to be true intoxications through putrefactive substances, ptomaines, etc. Recent investigations, however, have shown that they are mostly due to certain specific bacteria, and then either following the introduction of the specific pathogenic bacteria themselves (infection), or following the introduction of the specific poisons produced by the bacteria (intoxication).

It is often a difficult matter to establish the true cause of a case of food poisoning, though, of course, it is easier when the cases occur in large numbers, especially among the inmates of barracks, prisons, boarding schools, etc., where the conditions of life are uniform and readily controlled. When but one or two cases occur in a private family, it is much more difficult to ascertain the true cause, and then only by means

of most painstaking investigation. Such an investigation, moreover, requires an exact knowledge of the mode of origin of these poisonings, for otherwise the subsequent investigation will be directed into entirely false directions. Besides, the bacteriological examination which is necessary in most instances, can yield successful results only if the suspected material (remains of the suspected food, stool or vomit of the patient, etc.) is at once sent, properly packed, to the laboratory.

The more important of the food poisonings due to bacteria, often occurring in extensive outbreaks, are the following: meat poisoning, fish and shell fish poisoning, and poisoning through cheese, ice cream, puddings, potatoes, and canned goods.

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Bacterial Food Poisoning

I

MEAT POISONING

In studying the poisonings due to the eating of meat it is well to divide these into three varieties:

1. Poisoning due to the eating of meat from diseased animals. (This form is usually associated with the bacillus enteritidis or bacillus paratyphi.)

2. Poisoning due to the eating of putrefied meat. (This is usually associated with bacillus proteus and bacillus coli.)

3. Poisoning due to "sausage poison." (This is produced by the anærobic bacillus botulinus.)

In the first variety, poisoning is produced even by freshly-killed meat; in the other two varieties, the meat acquires poisonous properties only after the animal has been slaughtered. Clinically the first two varieties of poisoning manifest themselves largely by gastro-intestinal symptoms, while the third variety is associated especially with symptoms referable to the central nervous system.

POISONING THROUGH DISEASED MEAT

Sepsis Intestinalis (*Bollinger.*) *Infectious Enteritis* (*Gaffky.*)

This is the most common of the meat poisonings,

and is due to bacteria of the typhoid-colon group (bacillus enteritidis and bacillus paratyphi *B*) which infect the animal during life. Although these germs in no way alter the meat, they multiply and give rise to poisonous metabolic products, so that the clinical symptoms usually set in after a brief period of incubation.

HISTORICAL

One of the first to draw attention to this variety of poisoning was Bollinger, who described such cases in 1876. Five years later, this author [1] was able to report on eleven large outbreaks, embracing over 1,600 cases. The symptoms were chiefly gastro-intestinal, but varied considerably according to the quantity of meat ingested, and on the individual susceptibility of the patients. In fact, according to Bollinger, the symptoms ranged in a complete scale from simple digestive disturbances, such as a little gastric catarrh, or perhaps a diarrhœa with vomiting, to severe febrile disturbances, such as those of enteritis, gastric fever, typhoid fever, dysentery, etc. Dividing the cases symptomatically, he distinguishes three groups, which, however, are not sharply defined one from the other. These are:—1, choleraic cases with profuse diarrhœas, 2, cases running more of a typhoid course, with a longer incubation than the preceding and with marked cerebral disturbances of various kinds, and, 3, cases beginning with choleraic symptoms but soon presenting more the symptoms of typhoid fever. If the disease

is severe, convalescence is slow, and the patient is left weak and emaciated. The duration of the illness in these cases is several weeks. In the milder cases, on the other hand, the disease only lasts a few days. In the fatal cases death usually occurs from the fourth or fifth to the tenth or eleventh day, rarely after that time. The autopsy findings are usually those of a gastro-enteritis. The lymph nodules of the intestine are very prone to involvement, ulceration of the intestine being of frequent occurrence. The mesenteric glands may be swollen, and so also the spleen, and there may be hæmorrhages into various organs. Occasionally the entire anatomical picture resembles that found in typhoid fever. The period of incubation depends on the amount of infected meat ingested, and may vary from 6 to 24 hours, to as much as a week or more. Two epidemic outbreaks of this form of meat poisoning are described by Bollinger as being especially interesting, as the symptoms resembled those of typhoid fever.

One of the outbreaks occurred in Andelfingen in 1841, and embraced about 450 patients who had come together at a musical festival. Ten of the patients died. In all probability the direct cause of the poisoning was some roast veal which had been eaten in large quantity. The symptoms were nausea, vomiting, foul-smelling diarrhœas which exhausted the patients, dysphagia, dilatation of the pupils, disturbances of vision, delirium, and slow convalescence with great prostration. It was found that persons who had not attended the

festival, but who had eaten *beef* supplied by the same butcher, also were affected, so that it is probable that the infecting virus had been communicated from one meat to the other while lying in the butcher's shop. Cooking the meat did not destroy the poison. The period of incubation varied from three to ten days.

The other interesting outbreak described by Bollinger occurred in Kloten in 1878. Like the preceding, this was associated with the eating of meat at a musical festival. Five hundred and ninety-one of those who took part in the festival, numerous persons who had eaten meat supplied by the same butcher, and finally, also, a number of persons in whom the source of infection could not be traced, a total of 657 persons, were thus poisoned. Of these six died. The direct cause was found to have been the eating of the meat of a seven-day-old calf which had been slaughtered either after death or while moribund. In several of the cases the symptoms set in as early as the first day; in most, however, there was an incubation period of from four to six days. It is interesting to note that those who drank wine freely either were not affected, or suffered only a mild attack. The symptoms began with depression, headache, pains in the limbs, constipation followed by diarrhœa. Toward the end of the first week the cerebral symptoms became less prominent, stools assumed more of a typhoid character, and in the severe cases there was often a roseola and a small papular rash. At the height of the disease there was quite constantly an enlargement of the spleen, and the

superficial lymph nodes, especially those of the groin, were frequently swollen. A point of considerable interest is the fact that the outbreak gave rise to 55 secondary cases. The autopsy findings were enlarged spleen, infiltration of Peyer's patches and of the solitary lymph follicles, also ulcers in the small intestine either in a state of granulation or already cicatrized. On the basis of the clinical symptoms, as well as of the anatomical findings, and in view of the fact that secondary cases occurred, most of the observers regarded the disease as typhoid fever. This opinion was shared by Eberth, who had the opportunity of examining a number of the cases post mortem. For a considerable time there was quite a difference of opinion as to the real nature of the outbreak. One set of observers held that it was a true typhoid epidemic; the other regarded it as an epidemic of meat poisoning. Bollinger believed that the outbreak was due to a peculiar infection which closely resembled and in fact was closely related to typhoid fever. He thought it might be regarded as a variety of that disease, and proposed the terms *sepsis intestinalis,* or septiform gastroenteritis. (See page 15.)

Numerous outbreaks of meat poisoning have been reported in the last twenty years. Ostertag [2] was able to collect accounts of 85 epidemics in the period 1880-1900, the larger part of which were reported from Germany. The total number of cases was over 4,000. There is no doubt, however, that the actual number is much greater, since not all cases, even when they occur

in considerable groups, find their way into the literature. Bollinger believes that many cases of disease giving the clinical picture of febrile jaundice (Weil's Disease) belong to the group of meat poisonings.

ETIOLOGY

Most of the outbreaks of this kind of meat poisoning have been traced to the meat of calves which had become septic from navel infection, or to the meat of cows which were slaughtered because they had been infected after calving, or were suffering from some peculiar intestinal or udder infection. Schneidemühl[4] has analyzed 61 large outbreaks reported from 1868 to 1898, with the following result. The total number of cases was about 5,000, of which 76 died. In 38 of the outbreaks the disease was traced to the meat of cows, in 15 to that of calves, in 3 to that of steers, in 3 to that of hogs, and in two outbreaks to horse meat. In the case of the cows, in 16 instances the animals were killed because of gastric and intestinal disease; in 12 instances, because of disease of the organs of parturition; in 3 instances because of disease of the udder; and in 3 instances because of foot and mouth disease. In the case of the calves, intestinal and joint disease were the main reasons for slaughtering the animals. According to these figures, therefore, the chief source of this form of meat poisoning is the meat of cows which have been slaughtered because of disease of the gastrointestinal tract, because of septic infection after calving, and septic infection of the udder. Next most

dangerous is the meat of calves which soon after birth manifest signs of gastrointestinal disease, or pyæmic infection of the joints (polyarthritis, phlebitis of the umbilical vein). In a case of this form of meat poisoning recently reported by Bryson,[55] a Scotch shepherd became ill three hours after eating some mutton. Investigation showed that the sheep had been ill for several days, and had therefore been slaughtered. This method of disposing of sick animals is said to be common practice among the Scotch shepherds, the meat being known as "braxy." The patient vomited repeatedly, had severe abdominal pain, cramps in the extremities and some retraction of the head. There was also a slight rise of temperature the following day. Within a week the patient had completely recovered.

From the above analysis it is apparent that the majority of these outbreaks of epidemic meat poisoning thus far reported have been due to what may be called "forced slaughtering," or "emergency slaughtering" of diseased animals, and allowing the meat to be marketed without proper medical control. It has been found that usually the severest attacks followed the eating of the raw meat, cooking destroying the infecting bacteria. As a rule, however, cooking does not destroy the toxin produced by these germs, though there seem to be some exceptions to this. Cases have occurred in which the severest attacks were associated with the eating of the cooked meat and of the resulting broth.

Most of the outbreaks occur in the summer, and in

many instances following the eating of sausages, pâtes, chopped meat, etc. These forms of meat are especially dangerous because in their manufacture viscera, such as lung, liver and spleen, are often employed, and it is well known that the infecting bacteria often localize in these in large numbers. Furthermore, in consequence of the long period which often elapses from the time of manufacture to that of consumption, the bacteria may have abundant opportunity to multiply and produce their toxin. (Van Ermengem.) As a rule, in contrast to what is observed in "sausage poisoning" (page 62) all of the meat from these infected animals is poisonous immediately after slaughtering; it may, to be sure, increase in toxicity as the result of further development of bacteria and consequent further toxin production. It is usually impossible to distinguish the meat of such diseased animals from that of normal animals either by appearance, smell, taste, or consistency. The same is true of the dishes prepared from such infected meat; they appear quite normal. For this reason the meat is usually purchased and consumed without question. In the very acute septic infections, the meat often shows such slight changes that unless one is very familiar with the course of the disease and with all its symptoms, very little is found by macroscopic examination.

BACTERIOLOGY

Our knowledge of the etiology of the above-described form of meat poisoning has been aided

greatly by the bacteriological investigations of the past twenty years. In all probability the exciting cause is the bacillus discovered by Gärtner, the so-called B. enteritidis, which is a member of the typhoid-colon group.

In 1888 Gärtner[5] investigated an outbreak of meat poisoning in Frankenhausen, in which, two to thirty hours after eating the meat of an infected cow, 57 persons became ill with symptoms of gastroenteritis. One of the persons died. The cow from which the meat was derived was found to have been slaughtered because of an intestinal catarrh. Gärtner was able to isolate B. enteritidis in pure culture not only from the infected meat, but also from the spleen of the fatal case. This organism is a freely-motile bacillus, ~~not stained according to Gram,~~ produces no indol in peptone solution, and ferments dextrose with the formation of gas. By feeding, and by subcutaneous as well as intraperitoneal inoculations of pure cultures, Gärtner was able to infect mice, guinea-pigs, rabbits, sheep and goats. Cats, dogs, and chickens proved refractory. The susceptible animals had fluid evacuations, and on post-mortem examination showed a marked inflammatory hyperæmia in the intestines and abdominal viscera. Frequently this was hæmorrhagic in character. The lungs showed lobular pneumonic areas, and there were hæmorrhages into the organs. The bacilli could be demonstrated both microscopically and culturally in the blood and internal organs. The B. enteritidis forms a poison which is not destroyed by heat; guinea-

pigs and rabbits inoculated subcutaneously or by mouth with cultures which had been sterilized by heat, showed the same symptoms of gastroenteritis as those inoculated with living cultures. In addition they showed various nervous disturbances, paralyses of the lower extremities alternating with spasmodic twitchings, symptoms which are to be regarded as signs of toxic action. An interesting point in connection with this case is the fact that the mother of the dead boy, who had nursed him, subsequently developed the same disease, although she had not partaken either of the meat or the soup of the infected cow. In other words, she had become infected from the discharges from her son.

An interesting outbreak is that reported by G. Arbuckle Brown,[56] of Partick, Scotland, in which twelve persons became ill after eating canned meat. Although no bacilli were found in the meat, the poisoning was probably due to Gärtner's bacillus, as the serum of the patients agglutinated known cultures of this organism in dilutions of 1-30 and 1-60. The poisoning in this case would appear to have been caused only by a toxin produced by the bacilli in the meat, the organisms themselves having been killed during the canning process, but the toxin remaining intact.

Gaffky and Paak[6] in 1885, *i.e.*, three years before Gärtner's discovery, had studied an epidemic of meat poisoning in Röhrsdorf, which was traced to the meat from a horse suffering from abscesses. Eighty persons were taken ill, one of whom died. The meat had been eaten in the form of sausage made from the flesh

and liver of the animal. The authors were able to cultivate a bacillus from the organs of animals inoculated with the sausage. This organism was subsequently found to agree in its important characteristics with the bacillus later described by Gärtner, and was pathogenic for animals in feeding experiments. Unlike Gärtner's bacillus, however, the boiled cultures proved innocuous.

In 1889 Neelson, Johne and Gärtner[7] studied an outbreak of meat poisoning in Cotta, near Dresden, which was traced to a cow slaughtered because suffering from a purulent inflammation of the udder. There were 126 cases, with four deaths. The authors were able to isolate a bacillus identical morphologically and culturally with the B. enteritidis, not only from the suspected meat, and the bone marrow of the cow, but also from the intestinal tract, blood, and spleen of two of the fatal cases. The bacillus was pathogenic for mice and guinea-pigs. The cultures, however, were rendered innocuous by boiling, and the same was true for the meat and the soup made from the same. In passing it may be said that the toxin from B. enteritidis does not always withstand boiling.

Very careful examinations were conducted by van Ermengem[8] in connection with an epidemic of meat poisoning which occurred at Moorseele in Flanders, in 1891. Eighty persons were affected, of whom four died. The outbreak was traced to the eating of roasted and boiled meat derived from two calves suffering from enteritis. In most instances the symptoms

commenced within a few hours after partaking of the infected meat, although in one of the fatal cases the interval was four days. Cultures made from the bone marrow of the tibia of one of the calves, and from the liver, spleen, and intestinal contents of one of the fatal cases revealed the presence of a bacillus which agreed in all particulars with Gärtner's bacillus. Cultures sterilized at 100° C. and even at 120° C. were still toxic and produced marked inflammatory changes of a hæmorrhagic character. Van Ermengem called attention to the similarity which B. enteritidis bore to the bacillus of swine plague and the bacillus of hog cholera.

Holst[9] studied an epidemic occurring in Gaustad, near Christiania, in 1891. This outbreak was associated with the eating of meat from a calf suffering from enteritis. There were 81 cases, with 4 deaths. Cultures from the spleen yielded a bacillus which was identical in character with that isolated at the Moorseele outbreak and with known cultures of B. enteritidis. The Gaustad bacillus produced toxins which withstood heating, but it was found that this property was rapidly lost with artificial cultivation.

An interesting outbreak of meat poisoning occurred in Rotterdam in 1892. This was traced to the meat of a cow which had been slaughtered in accordance with the regulations at the municipal abattoir, and which had passed the prescribed inspection as normal. Poels and Dhont[10] were able to isolate a bacterium from the meat, and found that the organism was pathogenic for mice, guinea-pigs and rabbits, giving rise to intestinal

catarrh, and paralysis of the lower extremities. Even the sterilized cultures were toxic. When small amounts of the bacillus were injected intravenously into cows transient fever, muscular twitchings, loss of appetite and fluid evacuations were produced. The meat from these animals, slaughtered four days later, contained no bacilli and was eaten without any injurious results whatever. Another cow was killed 20 minutes after such an inoculation. Small numbers of the bacilli were found in the spleen, liver and blood. After the meat had been kept for three days at 20° C., however, the number of bacilli present was much greater. Some of the meat had been kept in the refrigerator, and contained but few bacilli. Fifty-three persons ate of this, and 15 were attacked 12 to 18 hours after with headache, colicky pains, and diarrhœa.

Basenau [11] examined the meat of a cow which had been slaughtered because of illness after calving. He isolated a bacillus which he termed B. bovis morbificans, which resembled the typhoid bacillus, was pathogenic in feeding experiments, and was killed by exposure to 70° C.

A small epidemic of meat poisoning occurred in Rumfleth in 1893. This was traced to the meat of a cow which had been ill for eight days after calving. The cooked meat as well as the broth seem to have been responsible for the poisoning. Fischer,[12] who studied the outbreak, isolated a bacillus from the meat and found it identical with B. enteritidis. Injected into animals it produced intestinal disturbances. Steril-

ized cultures also killed animals and produced similar disturbances. It was found, however, that the toxicity rapidly decreased with artificial cultivation of the organism. In 1895, in Haustedt, Fischer isolated the same organism from the meat of an ox which had been slaughtered because suffering from diarrhœa. The meat of this animal produced symptoms of poisoning in 50 persons.

In 1894 an outbreak of meat poisoning occurred in Bischofswerde, Saxony, almost 100 persons being affected. The cause of the poisoning was traced to sausages and chopped meat, composed of pork and beef. There were no deaths. Johne [13] examined the meat and isolated a bacillus closely related to the B. enteritidis.

In Ghent, in 1894, twelve persons became ill after eating a species of cervelat sausage which is eaten raw. The inspector having charge of the abattoir was asked to have an examination of the sausage made, but, saying that there was nothing wrong with it, cut off and himself ate a few slices and gave several to his assistants. The latter soon showed signs of a more or less marked enteritis; the inspector himself was attacked with severe choleraic symptoms, accompanied by albuminuria, diarrhœa, vomiting, and collapse, and died on the fifth day. Autopsy showed a very severe hæmorrhagic and gangrenous gastroenteritis, fatty degeneration of the liver, acute interstitial nephritis, etc. Van Ermengem [14] examined the sausages bacteriologically, and found, especially in the uneaten

part of the sausage cut up by the inspector, a very virulent and toxic bacillus which proved to be identical with B. enteritidis. The same organism was isolated from the blood, intestine, and other organs of the inspector. In this outbreak an instance of direct transmission of the disease was encountered. The husband of a woman who had become ill after eating some of the sausage, himself became affected with the same symptoms, although he had not eaten any of the sausage.

In recent years a number of observers have reported cases of epidemic meat poisoning in which bacilli were found identical with B. enteritidis, or at least closely related to that organism. The more important of these are given in Table I. (See page 30.)

In the majority of these instances the infected or suspected meat was derived from diseased animals after "forced slaughtering." Furthermore, it was repeatedly found that the bacteria were present not merely in certain particular organs, but also in the entire muscular tissue, and especially in the glandular organs. It was not always possible to demonstrate the bacilli in the suspected meat, for often there was no material left to examine. However, the bacilli were isolated from the stools of the patients and from various organs in the fatal cases. In the latter, they seemed to be especially plentiful in the spleen.

OCCURRENCE OF THE BACILLI IN NATURE

But little is thus far known concerning the occur-

TABLE I.

AUTHOR	LOCALITY	YEAR	CAUSE	NUMBER OF CASES
Flügge-Kaensche [15]	Breslau	1893	Chopped meat from cow slaughtered because of enteritis and liver disease	80. No deaths
Scheef [16]	Horb	1896	Liver sausage	50. No deaths. (B. Enteritidis Bucoli and Streptococci)
Günther [17]	Posen	1896	Pork	
Silberschmidt [18]	Canton Thurgau	1896	Boiled, corned, and smoked pork. (Corning and smoking does not kill the bacilli)	7. One death
Pouchet [19]	France. Département du Nord	1897	Meat pâte made from pork at a time when swine pest was prevalent	36. One death
Durham [20]	Hatton	1898	Veal pâte	
Durham [20]	Chadderton	1898	Pork	
de Nobele [21]	Aertryk	1898	Meat from a calf ill with severe enteritis	
Hermann and van Ermengem [22]	Sirault	1898	Veal	
de Nobele	Brügge	1899	Pork sausages	
van Ermengem	Meirelbeck	1899	Meat from a cow	
Trautmann [23]	Düsseldorf	1901	Horse meat	57.
v. Drigalski [24]	Neunkirchen	1903	Ham roll and sausage made from horse with suppurating processes. Meat had not been condemned by inspector	More than 30. Three deaths
B. Fisher [25]	Kiel	1903	Probably meat	Over 80.
Alsfeld	Greifswald	1904	Probably veal	50.
Uhlenhuth [26]		1905	Meat from healthy animal. At first eaten without harm. After being warm, became infectious in 24 hours	8.
Curschmann [27]	Berlin			
	Bern	1906	Chopped beef	90. Two deaths
Kutscher [28]	Giessen	1906	Liver sausage	26. Four deaths
Heller [29]		1906	Ham	32.
Fromme [30]				

rence of B. enteritidis and B. paratyphi* outside of the infected organism. They do not appear to be widely distributed. We also know nothing as to how these bacilli get into the animal. Levy and Jakobsthal [89] demonstrated the presence of true typhoid bacilli in a splenic abscess of a steer, so that true typhoid bacilli may perhaps also have some relation to meat poisoning. In a number of instances, the exciting organism was found in the organs of the slaughtered animals (Aertryk, Meirelbeck), or in their flesh (Breslau, Neunkirchen, Berlin, Giessen), showing that the animals had already been infected during life. In other instances, however, the meat was originally sound and was infected subsequently. Sound meat is easily infected by contact with meat containing these bacilli, especially if the pieces are laid one on the other. Infection may also result from infected persons handling or preparing the meat, for now we know that "bacilli carriers" are not at all uncommon. In studying outbreaks of meat poisoning connected with restaurants, canteens, and the like, the existence of such carriers must not be forgotten, and bacteriological examinations of the stools of such suspected carriers should be undertaken.

In order to gain some idea as to the occurrence of paratyphoid and enteritidis bacilli, Dieudonné recently made some examinations among the cattle, calves, and hogs killed in the Munich municipal abattoir. He selected those which, for one reason or another, had been sent to the sanitary division for further examination,

*For the relation of paratyphoid to enteritidis see p. 35.

especially animals with septic processes, such as endometritis, purulent peritonitis, suppurative inflammation of joints in calves, etc. Recent investigations having shown that typhoid and typhoid-like bacilli often are especially numerous in the bile, cultures of this were made at once and after enriching for 24 hours in the incubator. The cultures were made on malachite-green agar and on Drigalski's medium. In only a few instances was there any growth in the cultures made at once, and this was due to B. coli, with here and there a streptococcus. In the cultures made after enriching the bile, a profuse growth was always obtained. Forty-two cases were examined; in thirty-six instances the growth was mainly streptococcus, which produced marked acidity in the medium, a few staphylococci, and bacillus coli. In four instances bacillus coli alone was present, and in two instances bacilli were obtained which culturally and biologically were identical with paratyphoid bacilli. In one of these the culture was obtained from a calf which had become septic from navel infection. Pericarditis and joint inflammation were marked. In the other case the animal was a cow showing a suppurative peritonitis, abscesses in the liver and spleen, and general sepsis. The bile of both these animals was mucoid in character, yellow, and quite thick, whereas in most of the other animals it was normal in color. Evidently we are here dealing with inflammatory phenomena with increased production of mucus. According to Pies[40] this favors the growth of typhoid bacilli. Cultures made from the

abscesses in the liver and spleen also yielded paratyphoid bacilli, but no bacilli were found in the flesh or blood of these animals. In abscesses in the other animals streptococci were constantly present.

Uhlenhuth [42] examined the intestines of apparently sound hogs killed in the Berlin abattoir. In 6% of his cases he isolated bacilli which it was impossible to differentiate either from paratyphoid type B, or from bacillus suipestifer. These bacilli, it should be said, are evidently very closely related, as can be seen from their serum reactions. Bacillus paratyphoid B. is also a near relative of the bacillus of mouse typhoid.

Similar investigations were undertaken by Morgan [57] in 1905. This author concludes: "1. There exist in the intestines of healthy animals organisms conforming morphologically and biologically to the enteritidis and paratyphoid A types. 2. These organisms, as regards their agglutination reactions, fall into three groups, namely, those of the B. enteritidis, Aertryk, or hog cholera type, those of the B. enteritidis, psittacosis * type, and those of the B. paratyphoid A, unknown type. 3. The nomenclature adopted by different observers is unsatisfactory, but is based on the principal differentiating criterion we at present possess, namely, specific virulence."

Hübener [61] has recently reported on the occurrence of paratyphoid and related bacilli and finds that not

* Psittacosis is a contagious disease of parrots, communicable to man, marked by pulmonary disorder and high fever. The organism is fully described by Nocard and Leclainche, 1898.

only do these occur in the intestines of normal hogs, but also occasionally in perfectly sound sausages, and in the excreta of healthy human beings. Rimpau [62] examined 50 healthy school children and 50 healthy orphans and found paratyphoid bacilli, type B., in three of the former and one of the latter. No infections were produced by any of these bacilli carriers.

These results are very interesting, for they show, as Kutscher and Uhlenhuth emphasize, that we must bear these diseases of animals in mind when considering the etiology and prophylaxis of human paratyphoid infection. Further investigations are very much desired concerning the relations existing between these diseases and human paratyphoid infections. Attention will also have to be paid to the milk of such infected animals, as has recently been demonstrated by Fischer.[41] Investigating an epidemic of some 50 cases exhibiting the clinical picture of a severe gastroenteritis, this author was able to isolate a paratyphoid bacillus from the stools of two of the patients, and from the meat, internal organs, and milk of two cows which had died of gastroenteritis. Since none of the meat had been eaten by the persons affected, the epidemic was ascribed to the drinking of the milk from the infected cows.

Mühlens[25a] has recently studied the occurrence of bacillus enteritidis, seeking especially to learn whether this organism was commonly present in food. Fifty-seven samples of different kinds of meat were examined—pickled goose breast, raw ham, pork, ox

tongue, corned beef, smoked sausage, smoked salmon, smoked herring, etc.—the samples were examined culturally, but no bacteria of this group were found. At the same time each of the samples was fed to two or three white mice, the greatest care being taken to prevent the animals from infecting one another. Forty out of the fifty-seven samples killed one or more of the animals fed. From seventy out of seventy-four such mice bacteria belonging to the " enteritis " group were isolated either from the spleen, the blood, or the intestines. It would seem that such bacteria may be present in very small quantity so as to be overlooked by cultural examinations. Under favorable circumstances the bacteria may multiply in the meat to a sufficient degree to become pathogenic for man, thus causing the disease known as " food poisoning."

BIOLOGY OF THE BACILLI

The cultural characteristics of the bacilli isolated in the various outbreaks are quite regularly similar, and correspond to those of the bacillus enteritidis and of the bacillus first described by Schottmüller [81] as paratyphoid, type B. These bacilli can be distinguished from typhoid bacilli and colon bacilli in a number of ways, but especially by their behavior to various sugars. This is well shown in Table II

From this it will be seen that B. enteritidis and B. paratyphi B. ferment glucose but not lactose; B. coli ferments both; B. typhi ferments neither.

An instructive table showing the biological charac-

TABLE II.

	TYPHOID	BACILLUS ENTERITIDIS AND PARATYPHOID, TYPE B	B. COLI
Potato	Delicate, fine, hardly visible pellicle	Grayish brown thick	Thick yellow or yellowish brown
Milk	Not coagulated; slight production of acid	Not coagulated; after two weeks clearer; alkaline, yellowish tint	Coagulated; marked acid production
Glucose agar	No gas formed	Gas formed	Gas formed
Neutral red agar	Not reduced	Fluorescent; gas produced	Fluorescent. Gas produced
Litmus whey	Clear, acid	At first acid, then alkaline	Cloudy, acid
Glucose-Nutrose solution	Acid production, coagulation	Acid production, coagulation	Acid production, rapid coagulation
Lactose-Nutrose solution	Unchanged	Unchanged	Acid production, rapid coagulation
Drigalski agar	Blue colonies	Blue colonies	Red colonies
Endo's agar	Colorless colonies	Colorless colonies	Red colonies
Malachite-green agar	Delicate growth, no decolorization	Vigorous growth, color yellow	Little or no growth
Indol reaction	No indol	No indol	Indol produced
Pathogenicity for mice and guinea-pigs	But little pathogenic	Markedly so Toxins withstand heat	But little pathogenic

teristics of the bacteria belonging to this group has also been prepared by Morgan.[57]

The best medium for isolating these bacilli is the malachite-green agar of Lentz and Tietz. On this B. coli grows little or not at all, B. typhi shows delicate green colonies without discoloring the medium, while B. enteritidis and B. paratyphi B. grow abundantly and change the color to yellow. This medium might therefore be called a selective one.

An important means of differentiation is afforded by the tests on animals, especially on mice and guinea-pigs. Typhoid and colon bacilli are but slightly pathogenic for these animals, whereas both enteritidis and paratyphoid are markedly pathogenic. In fact, the latter two organisms produce a toxin which when given per mouth kills susceptible animals with symptoms of gastroenteritis. This toxin, moreover, is generally quite resistant to the action of heat, though here there is some individual variation in the different cultures. This explains what has already been said, that infected meat, whether roasted or boiled, and even the broth made from such meat, may cause severe toxic symptoms. When the bacilli are cultivated for some time on artificial media, the virulence rapidly decreases, and so does the production of the resistant toxin.

TECHNIQUE OF THE BACTERIOLOGICAL EXAMINATION

The isolation and identification of the exciting cause of an outbreak of meat poisoning is quite a difficult matter, and can be successfully performed only by

trained laboratory workers. It is extremely important, therefore, that the required material be properly collected, be as fresh as possible, and then be properly sent to the laboratory. The material to be examined will include the suspected meat (sausage, pâte, etc.), vomit and stool from the patient, the patient's blood, and in case of autopsy, pieces of the intestine, spleen, and liver.*

According to Basenau [11] it is well to wait with the bacteriological examination of the meat until 24 hours after slaughtering. In that time the bacilli have multiplied, even at the low temperature at which the meat is kept, so that the bacteriological examination is simplified. It is well to select a piece of meat rich in loose areolar tissue. The surface is seared with a hot iron, and then an incision is made through the seared area with a sterile scalpel. Smears and cultures are thus made from the inside of the meat. The cultures are made by smearing some of the material with a fine platinum loop over the surface of gelatine and agar plates, over plates made after the method of Conradi-Drigalski, and over malachite-green plates. In preparing these plates it is well to plate various quantities of a suspension made from the inside of the suspected meat. Another portion of the suspected meat may be

*The various materials should be placed each in a wide-mouthed sterile bottle tightly corked, the bottles packed with ice and saw-dust, and sent at once, by special messenger if possible, to the nearest laboratory. All the bottles should be carefully labeled as to their contents, the date of collection, and a complete clinical account should be sent to the bacteriologist for his guidance.

kept for 24 hours at room temperature so as to "enrich" it, *i.e.*, in order to give the bacteria time to multiply. After this, smears and cultures are made as with the original specimen. In addition to this it will be well to feed some of the meat to mice. Basenau [11] advises that two mice be fed with the meat raw, and two mice with meat which has been heated for one hour to 100° C. Mice are particularly well suited to such feeding experiments because of their extreme and uniform susceptibility. The vomited material and stool are sown on plain agar, on Drigalski medium, and on malachite-green plates. The last named is very highly spoken of for these examinations, as it also inhibits the growth of B. coli. Some of the stool is used for inoculating mice subcutaneously. The examination of the patient's blood is of considerable diagnostic significance. The most convenient method is to draw from 5 to 8 cc. by means of a sterile glass syringe and needle from a vein at the bend of the elbow. It is unnecessary to make any incision. The skin is to be cleansed, compression of the upper arm is made by an assistant, and the needle is thrust directly into the distended vein. The blood is distributed among a number of tubes of melted agar, and also planted into broth. Inoculation into bile medium has given good results in the hands of several observers. A small quantity of blood may be left to clot in the syringe, the serum which separates being subsequently used to make agglutination tests.

The bacteria isolated from the infected meat, from the fæces of the patients, and from the body of the

fatal cases must be tested by all known methods of differentiation, including biological properties, pathogenicity, and serum reactions. Of these the last named is the most delicate. The bacilli should be tested with the serum of an animal immunized against known culture of B. enteritidis or B. paratyphi B. In this way it is possible to differentiate these organisms from typhoid or colon bacilli. Absorption tests should also be undertaken.*

Extensive investigations made by de Nobele,[21] Trautmann,[33] Uhlenhuth, and others have shown that the bacteria of meat poisoning may be divided into two types. According to Uhlenhuth [26] these are as follows:

GROUP I.

B. enteritidis (Gärtner) B. Moorseele (v. Ermengem) B. Ghent (v. Ermengem) B. Brügge (v. Ermengem) B. Rumfleth (Fischer) B. Haustedt (Fischer)	B. enteritidis, Gärtner, group

GROUP II.

Paratyphoid B. B. Breslau (Flügge-Känsche) B. Meirelbeck (de Nobele) B. Düsseldorf (Trautmann) B. Sirault (Hermann & v. Ermengem) B. Aertryk (de Nobele) B. Neunkirchen (v. Drigalski) B. Greifswald (Uhlenhuth)	Paratyphoid B. group

* The principle of these is discussed in Bolduan, "Immune Sera," Wiley & Sons, N. Y.

Bacilli of the paratyphoid B. type were also found in meat poisonings in Alsfeld (Curschmann), in Berlin (Kutscher), in Bern (Heller), and in Giessen (Fromme).

CLINICAL TYPES OF CASES

According to van Ermengem [8] the clinical course of the disease seen in most of the reported outbreaks varied considerably. Gastrointestinal symptoms, however, were almost always prominent. The attack usually manifested itself like one of cholerine, or cholera nostras, or of inflammatory gastroenteritis. In addition to the main symptoms, offensive, yellow, diarrhœal evacuations, colicky pains, vomiting, and muscular weakness, there are frequently albuminuria, catarrhal pneumonia, and various cutaneous lesions, such as herpes, polymorphous erythema, roseola, urticaria, scurvy-like hæmorrhages into the skin, or petechiæ. Dilatation of the pupils, and photophobia have been observed in some cases, and occasionally there has been marked scaling of the epidermis on the inner aspect of the hands and soles of the feet. In most instances, symptoms began in from six to twelve hours after the meat was eaten; occasionally, however, the interval was much longer. In a few cases, the vomiting and diarrhœa began immediately after the meal, just as in an ordinary indigestion. In general it may be said that the severity of the attack depends on the amount of meat eaten. The mortality varies from 2 to 5%. At the autopsy one usually finds more or less well-marked signs of gastroenteritis, often hæmorrhagic in

character. The solitary lymph follicles and Peyer's patches are swollen and prominent; frequently there are ulcers both in the small and the large intestine. The spleen is enlarged, and the kidneys and liver are congested. In cases which have run a rapidly fatal course, there are usually no distinct anatomical changes.

Trautmann,[88] struck by the close biological relationship evidently existing between B. enteritidis and the paratyphoid bacillus, called attention to the relation existing between the respective diseases produced by these organisms, *i.e.*, between epidemic meat poisoning and paratyphoid fever. In meat poisoning, as we have seen, certain clinical as well as pathological features are distinctly typhoidal in character. Since both diseases are produced by the same organism, Trautmann regards meat poisoning as a highly acute, and paratyphoid fever as a more subacute form of a common infectious disease. His explanation for the occurrence of these two forms is as follows: In meat poisoning the slaughtered animal is infected, and when the meat is eaten both the disease germs and their toxic products poison the patient. The short period of incubation in meat poisoning, as compared with that in typhoid and paratyphoid, is therefore due to the large number of germs introduced, and to the fact that these are accompanied by their toxic products. If the amount of poison is too much for the body and if the bacteria invade the body juices, illness and death follow. In paratyphoid the period of incubation, *i.e.*, the time during

which the bacilli multiply sufficiently to produce toxic symptoms, proceeds in the body of the infected human. The bacilli develop slowly, and gradually give rise to symptoms of illness. Even in meat poisoning the disease may take a typhoidal course. This will occur when but few bacteria and little toxin have been ingested. In the pathological findings there is considerable similarity between paratyphoid fever and cases of meat poisoning running a prolonged course. Both exhibit the same lack of strict localization of the intestinal changes, both the same hæmorrhagic character, and the same main symptoms. Another point of agreement is the remarkably low mortality despite the severity of the clinical symptoms. According to Kayser [34] the reason for the short incubation period in meat poisoning as compared to paratyphoid is to be found not only in the introduction of preformed toxins, but in the fact that the path of infection differs. Typhoid and paratyphoid fever he regards as primarily a lymph and blood infection; the intestine is affected secondarily. In meat poisoning, on the other hand, the bacilli reach the intestine at once, and multiply there.

Kutscher,[38] moreover, calls attention to the fact that in paratyphoid infections not due to meat poisoning one often sees cases exhibiting entirely the clinical picture of a severe cholera nostras. Cases of this kind have been described by Schottmüller,[31] and by Hetsch.[35] The latter observed a large epidemic of paratyphoid in Kottbus in 1905, and noted that most of the cases ran a choleraic course. Rolly [36] divides the paratyphoid

infections according to their clinical course, and recognizes two groups of cases, namely, cases presenting the symptoms of an ordinary typhoid fever, and cases of a gastric type, in which gastrointestinal symptoms are prominent. He describes a case of the latter type which ended fatally, and exhibited the clinical picture of cholera nostras. This division of the cases corresponds, as can be seen, with that made by Bollinger in his meat poisonings in 1881. Trautmann states that many of the cases of meat poisoning seen in the epidemics in Andelfingen and in Kloten corresponded entirely to cases of paratyphoid infection. The similarity is still more striking when it is recalled that in these epidemics instances of secondary infection occurred in persons who had not eaten any of the infected meat. These were probably due to contact infection. In the Kloten epidemic the number of secondary cases was 55. We see, therefore, that these more recent etiological and bacteriological investigations confirm the view expressed by Bollinger [1] in 1881, namely, that these forms of epidemic meat poisoning are closely related to typhoid fever. Zupnik [87] speaks of those varieties of meat poisoning which run a more or less marked typhoid course as "typhoidal meat poisoning."

DIAGNOSIS

As a rule the diagnosis of this form of meat poisoning is extremely difficult unless a number of cases develop almost simultaneously. The cases with a long period of incubation are especially liable to be

overlooked. In any event the diagnosis will rest mainly on the history of the case.

SERUM DIAGNOSIS

The first to employ serum diagnosis in cases of epidemic meat poisoning was Durham,[32] who investigated an outbreak in Hatton. From the body of a person who had died after eating a meat pâte Durham isolated a bacillus resembling the B. enteritidis, and found that it was agglutinated by the serum of the other persons affected and of the convalescents in dilutions of 1 : 100 to 1 : 1000. In subsequent outbreaks similar observations were made. The bacillus isolated from the suspected meat or from the organs of the fatal cases was agglutinated by the serum of the convalescents in dilutions of 1 : 500 to 1 : 1000. The technique of the serum test is exactly the same as that employed in the Widal test for typhoid.* Whenever possible the serum of the patients and of the convalescents should be tested with a reliable laboratory culture of B. enteritidis or of B. paratyphi B.

It is possible in this way to establish a diagnosis of epidemic meat poisoning even after the outbreak has passed, for the agglutinating property persists in the blood of the patients for several weeks.

It may be well to say that the bacillus isolated is not always agglutinated in as high a dilution as a heterologous organism of a closely related species. Thus, in

* See in Rostoski-Bolduan "Serum Diagnosis," Wiley & Sons, New York.

a careful investigation of this question in connection with an outbreak due to B. enteritidis, Liefmann [58] found that the sera of the patients agglutinated typhoid bacilli in a higher dilution than the enteritidis organism isolated from the stools of the patients. There was conclusive evidence, however, that the latter was the cause of the infection.

PROPHYLAXIS

The prevention, or at least diminution, of this form of meat poisoning can only be effected by a properly organized system of meat inspection, especially in all cases of "forced" or "emergency slaughtering." Such an inspection must embrace a careful and thorough examination of all the organs by a competent veterinarian. It is essential that all meat which might give rise to disease be excluded from sale for human consumption. This will include especially the meat of calves and cows which have been slaughtered because of septic infection, and meat in which abscesses are found in the interior of muscles. Moreover, since the meat often shows no macroscopical changes, it will be well to adopt the suggestions of Basenau, Ostertag, v. Drigalski, and others, and resort to bacteriological examinations in all cases where there is any suspicion that the meat may be infected. Such examinations could be made either in a special laboratory connected with the abattoir, or in one of the regular hygienic laboratories. In case infection were demonstrated by this examination, the meat would of course be con-

demned and destroyed. Ostertag believes that such a laboratory, would prove an economic advantage, for if the suspected meat proved free from infection, it could be passed for market, whereas at the present time it is customary to order it condemned, in order to be on the safe side.

TREATMENT

The treatment of these meat poisonings will naturally depend on the type of case one is dealing with. In the acute cases, with a short period of incubation, it is well to wash out the patient's stomach, and this plan should be followed even though the patient has vomited. During the onset a brisk cathartic, such as castor oil or calomel, should be administered. Later, the administration of stimulants, especially alcohol, is indicated. Alcohol seems to exert an inhibitory action on the poison, and has given good results in a number of reported cases. Gastric irritability is treated with sedatives, mucilaginous drinks, cracked ice, heat to the epigastrium, etc. The diarrhœa is best treated with the initial dose of castor oil, followed by opium, rectal infusions, etc. The typhoidal cases are treated exactly like cases of typhoid fever, and the same precautions should be observed to prevent infection of others.

II

MEAT POISONING—(CONTINUED)

POISONING DUE TO EATING DECAYED MEAT

ETIOLOGY

THIS variety of poisoning is due to the eating of meat from healthy animals, which originally was sound, but which subsequently acquired toxic properties, owing to the introduction of putrefactive organisms. This form of decomposition may be due to any of the numerous putrefactive bacteria; as a rule, however, the majority of cases are due to the members of the proteus group of organisms and to the colon bacillus. The latter was isolated by Fischer [12] in cases of meat poisoning, once from a liver pâte, and once from some liver sausage. The colon bacillus produces a strong toxin which withstands heat.

This form of meat poisoning is observed chiefly after the ingestion of chopped meat, for this, almost invariably, is rich in germs. Sausages and game are also responsible for a large number of cases. Putrefactive decomposition of meat, however, does not invariably give rise to symptoms of poisoning. This is shown, for example, by the extensive consumption of game which is a little "high." Which of the putrefactive products is especially poisonous, is impossible to say. Further researches in this direction are much

desired. In all probability the poisoning is due to specific toxins produced by members of the proteus group of organisms. These toxins are destroyed by heat.

Van Ermengem[3] states that these meat poisonings are much less common after the ingestion of meat from healthy animals which has become infected after killing than after meat from diseased animals similarly infected. Most of these poisonings are observed in summer, probably because the bacteria can more readily multiply in the temperature then prevailing outdoors. In many instances the cases were due to the eating of chopped meat. This is often mixed with water for purposes of deceit, and this admixture favors the multiplication of the bacteria.

SYMPTOMS

Clinically this form of poisoning exhibits usually the picture of an acute gastroenteritis running a rapid course, and, as a rule, without fever. The severity of the symptoms depends on the amount of meat eaten, and on the age and resistance of the patient. In most instances the symptoms appear in from 4 to 20 hours after eating the meat, and consist in vomiting, headache, dysenteric, foul-smelling stools, colicky pains, weakness, etc. In more severe cases there may be convulsions, pains in the back and neck, and great depression. Most of the cases, even the severe ones, terminate favorably, though a feeling of weakness may persist for some time. Deaths are infrequent.

BACTERIA FOUND IN VARIOUS OUTBREAKS

According to statistics compiled by Schneidemühl,[4] outbreaks of this form of meat poisoning were observed in 1879 in Chemnitz, where 241 persons became ill after eating raw meat and sausage. Some of the cases ended fatally. In 1886, 160 persons became ill in the same city after eating raw chopped meat. The meat was prepared during a spell of very hot weather, and had been kept for some time. It had, however, been derived from healthy animals. The same meat eaten roasted or boiled was either entirely innocuous or at the most produced only transient malaise. Haupt found that this outbreak was due to a variety of the bacillus proteus.

In 1887, 20 persons became ill in Plauen after having eaten raw chopped meat which had been prepared six days previously and was in a state of beginning decomposition. A similar outbreak is reported from Gerbstadt in which 50 persons became ill after eating raw chopped meat, and several kinds of bologna sausage.

An interesting outbreak is described by Levy,[44] who observed bloody diarrhœa and vomiting in 18 persons frequenting a certain saloon. It was the custom here to keep the meat from day to day in a refrigerator which was found to be covered with a slimy brown crust having a disagreeable sour smell. From the bottom of the ice box proteus bacillus was isolated in pure culture. The meat stored in the box had become

infected with proteus and had been the cause of the outbreak. One of the cases ended fatally. At the autopsy the bacteria were readily found in the intestinal contents, but not in the blood of the individual. When pure cultures of this organism were injected into animals, the clinical symptoms produced resembled closely those seen in the persons attacked during the outbreak. The organisms apparently did not multiply in the animal body. Levy therefore believes that the pathogenic action is not an infection, but an intoxication, the proteus splitting the albumins and thus producing a poison.

In 1897 Wesenberg [45] observed an outbreak of meat poisoning in Mansfield, in which 63 persons were affected after eating chopped meat. The meat was derived from a cow slaughtered in an emergency. All who had eaten the meat boiled or roasted remained well. The persons affected had eaten the meat raw. There were no deaths. Examination of the meat showed the presence of proteus bacillus which was highly virulent for test animals. It was shown that the meat became infected after killing; it had been stored in a damp close cellar, and instead of hanging free on hooks, had been piled together, piece on piece.

Glücksmann [46] reports the case of father and son who became ill after eating from a piece of half-smoked pork. The father died. Other persons, who had eaten the same meat, but boiled or roasted, remained unaffected. Examination of the smoked meat showed the presence of proteus vulgaris.

Silberschmidt[47] described an outbreak of meat poisoning affecting 44 persons, one of whom died. The outbreak was traced to the eating of smoked sausages in which large numbers of proteus bacilli were found. While the smoking had not killed the bacilli, it sufficed to hide the disagreeable odor and taste produced by these organisms. Mice and guinea-pigs were killed by feeding the infected sausage; proteus could not be isolated from the organs of these animals, but was found in the intestinal contents.

In 1900 an outbreak of meat poisoning occurred among the troops in Hannover. According to A. Pfuhl,[48] who observed this outbreak, 81 soldiers were attacked with symptoms of acute gastroenteritis, which, however, soon subsided. The poisoning was caused by a peculiar kind of sausage which, instead of being filled in sausage cases, is packed into jars, making a kind of head cheese. In color, taste, and odor the sausage appeared perfectly normal. The bacteriological examination, however, disclosed the presence of bacillus proteus.

In the following year in the same city 34 persons became ill several hours after eating this same kind of sausage. The chief symptoms were nausea, profuse diarrhœa, repeated vomiting, and considerable depression. In most of the cases the symptoms subsided within 12 hours. The sausage was examined by Schumburg,[49] who isolated a variety of proteus. When the sausage was fed to mice and rats, the animals died after 24 hours with severe intestinal disturbances.

Proteus bacilli could also be isolated from the organs of these animals. Mice and rats fed with pure cultures of the organism died with symptoms of severe enteritis.

All observers agree in believing that the meat involved in these outbreaks was originally sound, and was derived from sound animals. The proteus infection takes place afterwards, probably because the meat is not properly kept. According to the investigations of Glücksmann, Silberschmidt, and others, the disease is not merely an infection with proteus bacilli, but also an intoxication with the metabolic products of these organisms. The proteus bacilli ingested with the food multiply in the intestinal tract and produce poisons which give rise to the constitutional symptoms. The intoxication is thus an accompaniment of the infection. A general infection, *i.e.*, a flooding of the body with bacteria, is probably very rare. This is indicated by the results of animal experiments. As was stated above, animals infected with proteus die with symptoms of severe enteritis, yet the bacilli are usually not found in the organs. Occasionally the proteus may already have developed its poisonous products in the meat. This, however, is not often the case, for the odor of these putrefactive products would at once excite suspicion (ammonia, hydrogen sulphide, indol, etc.). In most of the outbreaks of meat poisoning belonging to this group, we are expressly informed that the infected meat or sausage showed nothing abnormal in taste, odor, or appearance.

While smoking does not kill the proteus bacillus, as

can be seen from the case reported by Silberschmidt, it may occasionally hide some little disagreeable odor or taste. Heating for half an hour, to 80° C., on the other hand, not only kills the bacillus, but also destroys the poison which it produces. In this respect the proteus poison differs from that produced by B. enteritidis and B. paratyphi.

An epidemic of meat poisoning following the eating of potted tongues has recently been reported by Berry.[62a] The outbreak occurred not far from Liverpool in the middle of January, 1908, and affected about 170 persons. The symptoms came on 36 hours after eating. In preparing "potted tongue" the tongues were first kept in brine for three or four days, then cooked, and canned while hot. Some of the cooked tongue in question was found to be in a bad state of preservation, and smelled badly. Bacteriological examination disclosed the presence of B. enteritidis and of an organism similar to B. coli. There was some evidence to show that infection of the tongues had occurred in the brine.

It may be mentioned, in passing, that Ohlmacher[59] investigated some cases of poisoning due to eating oatmeal, and isolated the bacillus proteus as the cause of the outbreak.

In addition to the colon and proteus organisms, meat poisoning may occasionally be produced by the hay bacillus (B. subtilis). An instance of this is reported by Lubenau,[50] who observed an outbreak among the inmates of the sanatorium Beelitz, in 1906. About

three-quarters of the 400 inmates and a large part of the help were affected shortly after eating a dish known as "Königsberger Klops," a kind of meat croquette. This had been served at the noon meal; at about 11 o'clock in the evening a large number of persons were suddenly seized with profuse diarrhœa, persistent vomiting, severe headache, and great prostration. In two or three individuals fever was also present. Most of the cases recovered within three or four days. In a few patients the diarrhœa persisted for several weeks. Bacteriological examination of the meat balls revealed the presence of a bacillus belonging to the hay bacillus group. Grown in milk, this organism produced a strong poison which was pathogenic for young dogs. The dogs suffered from bloody diarrhœa and vomiting, and showed a marked loss of weight. The organism, called B. peptonificans, belongs to that group of hay bacilli which produces peptone in milk, and which has been held to be associated with the intestinal disturbances of infants. Investigation showed that the meat balls had been prepared from meat which had been kept in the refrigerator for four days. Then, since it was still fresh, it had beeen parboiled and kept for two more days before using. The boiling had not sufficed to kill the resistant spores of the bacilli, and the temperature in the refrigerator during the last two days had been sufficiently high to allow the spores to develop.

In view of the extensive distribution of the proteus bacilli, it is curious that this variety of meat poison-

ing is relatively infrequent. Very likely, however, it is much more frequent than the reports indicate, the rapid course of the poisoning often causing the attack to pass without its nature being recognized. It is certain that the meat of animals slaughtered in an emergency is much more to be feared than the meat of sound animals. According to Bollinger four-fifths of the meat poisonings are due to the former. The meat of diseased, especially of septic animals, decomposes very readily, and is dangerous even when only a slight degree of putrefaction has taken place. It is probable that the formation of poisonous products which has begun during life, continues after the meat has been dressed. As a rule the more rapid its putrefaction, the more poisonous is the meat.

Owen [60] has recently reported on an interesting outbreak of meat poisoning which occurred in Kalamazoo, Michigan, in 1906, and which embraced nineteen cases. Within a few hours after eating the meat, the patients complained of rapid pulse, fever, and severe abdominal pains, with vomiting and purging. Several had muscular cramps in the legs; one had convulsions. All the patients made a good recovery in a few days. The suspected meat was dried beef, all obtained from the same butcher, and probably infected in his shop. The meat had a normal odor and appearance, except for a slight greenish tinge often seen in sound meat of this character. Emulsions of the meat were fatal when injected into rats. Careful bacteriological examination revealed a staphylococcus as the probable cause of the

poisoning. This organism was isolated, practically in pure culture, from along the fasciæ, but not in the rest of the meat.

Although no bacteriological examinations were made, the following recent outbreak is reported because it suggests still another source of bacterial infections. In a hospital in New York, fourteen out of thirty-eight nurses suddenly became ill three or four hours after eating some minced chicken. The symptoms were nausea, vomiting, prostration, diarrhœa. All of the patients recovered by morning and were able to attend to their regular duties. The chickens used for preparing the minced chicken had been bought as fresh-killed, though it is probable that some, at least, were cold-storage fowls. They were cooked on Saturday, and the resulting soup was eaten by patients and others without producing any symptoms. Taken out of the soup caldron, they were kept, whole, in the refrigerator until Sunday afternoon, when they were cut up, minced, mixed with chicken "stock" and spices, and served as minced chicken for the evening meal.

The rapidity with which the symptoms developed point to a preformed poison as the cause of the outbreak. It is possible that chickens kept in cold storage for some time become poisonous owing to the bacterial changes going on even at that temperature.*

* The extent of bacterial growth which can take place at 0° C. is but little appreciated. This phase of the subject has recently been studied by Pennington, who finds, for ex-

We know from the investigations conducted in the U. S. Department of Agriculture by Pennington [56a] that macroscopically visible degeneration does occur under these conditions. Few bacteriological investigations have been made in this direction,* so that at the present time it is impossible to say just what rôle, if any, cold-storage chickens play in the causation of food poisoning. It may not be out of place to call attention to the fact that the cold storage of *undrawn* chickens, game, etc., is not at all comparable to the keeping of dressed meats, such as beef, veal, pork, mutton, etc., under similar conditions.

DIAGNOSIS

The diagnosis can only be definitely established by means of bacteriological examination. For this purpose plates should be made from the suspected meat, and animals, preferably mice, infected by feeding. When the poisoning is caused by bacillus proteus, the animals usually die in twenty-four hours with symptoms of gastroenteritis. The germ can then be isolated from the intestinal contents. It is useless to examine the meat chemically for the presence of ptomains,

ample, that very clean milk containing originally 300 germs per cc. when kept for 5 or 6 weeks at a temperature slightly less than 0° C., contains several hundred million germs per cc. The taste and odor of the milk is not changed by this enormous bacterial development, nor does the milk coagulate on heating. (See Journal of Biological Chemistry, Vol. IV, 1908, page 353.)

* Brown, H. R., 39th Annual Report, Mass. St. Bd. Health.

putrefactive alkaloids, etc., as such an examination yields no conclusive information. The only one competent to properly deal with this material is a trained bacteriologist.

EVIDENCES OF PUTREFACTION

It is sometimes difficult to recognize beginning putrefaction. The process commences at the surface and then spreads to the deeper portions. At first the connective tissue is affected, and extension is usually along the fibrous tissue, especially in the neighborhood of a bone or a large vessel. (Schneidemühl.[4]) As the process continues, a thick greasy-looking layer is formed and a gradually increasing foul odor is given off. When this has continued for some time, and connective tissue and muscle have completely disintegrated, putrefaction is complete. The cut surface of such meat appears porous, indentations made by the finger persist, the fat, originally yellow, is greenish, the bone marrow is soft, or even fluid, and has a greenish or brownish tint. The foul odor is most marked about the bones and in the fat. This odor is not lost by boiling or roasting the meat.

In examining meat for evidences of putrefaction it is well to first examine the reaction of the meat to litmus, and so seek to discover the presence of some of the products of putrefaction, such as ammonia. For this purpose it suffices to lay a piece of litmus paper on a freshly cut section of the meat. This test is not always reliable, for corned beef and smoked ham give

an alkaline reaction even when perfectly sound. Furthermore, the alkaline reaction of putrefaction may be masked by the acid products of a fermentation going on at the same time. It is better, therefore, to test for the presence of free ammonia according to the method devised by Eber. Into a test tube about five inches in length, pour about half an inch of a mixture composed of one part pure hydrochloric acid, three parts alcohol, and one part ether. The mixture is shaken and then a clean glass rod, rubbed into the suspected meat, is quickly thrust into the test tube, so that the lower end of the rod is about half an inch from the surface of the fluid. In the presence of ammonia one at once sees gray, bluish, or whitish fumes form about the end of the rod and sink to the surface of the reagent. This test is also not entirely free from objections, for sound corned meat may give a positive reaction owing to the frequent presence of trimethylamin. However, in the presence of other signs of putrefaction, this test serves to confirm the diagnosis. In important cases a bacteriological examination should always be made.

PROPHYLAXIS

In the prophylaxis of this form of meat poisoning, particular attention must be paid to the manner of keeping the meat. When proper facilities for cooling are not available, it is important to eat the meat as fresh as possible. Chopped meat is especially dangerous. Meat showing evidences of putrefaction should not be eaten. Boiling and roasting or frying the meat

lessens the danger. Special care should be exercised in summer, when most of these outbreaks have been observed. In badly constructed refrigerators putrefaction may readily occur. Owing to the lack of ventilation and the high degree of moisture, the putrefactive bacteria may develop in large numbers. It will be recalled that in the outbreak reported by Levy, the B. proteus was isolated from the bottom of the refrigerator. In order to prevent putrefaction, the refrigerators should frequently be thoroughly cleansed with hot soda solution. Keeping meat in refrigerators poorly or not at all iced is highly dangerous. Education of the public on this subject, and rigid inspections of the meat markets by the health authorities are necessary to prevent this form of meat poisoning. Chopped meat should be especially well scrutinized. In view of what has been said above, it would appear advisable to establish a maximum time limit beyond which undrawn poultry could not be kept, even in cold storage.

TREATMENT

The treatment of this poisoning is symptomatic, and consists in washing out the stomach, securing free evacuation of the bowels by means of cathartics, and administering stimulants if necessary. Alcohol appears to be especially useful. In general the treatment is that outlined on page 47.

III

MEAT POISONING—(CONCLUDED)

SAUSAGE POISONING (*Botulism, Allantiasis*)

HISTORICAL.

THIS group of meat poisonings, characterized by severe nervous symptoms, is usually spoken of as "sausage poisoning," because most of the cases reported followed the eating of sausages. Other forms of food have, however, been implicated, and the causative bacterium, the B. botulinus, was isolated during an outbreak due to poisoning by ham. This bacillus is a strict anærobe, and the poisonings are therefore observed in connection with foods which have been kept hermetically sealed or at least closed so that air is mostly excluded. Furthermore, it is usually found that the implicated articles have been eaten without previous cooking. This includes particularly sausages contained in thick sausage skins, to meat pâtes thickly embedded in fat, to pork insufficiently corned, etc. In most cases the meat used in the preparation of these dishes has been found to have come from perfectly sound animals. Other cases of this form of poisoning have been traced to the eating of canned fish and canned beans.

The first definite account of sausage poisoning was published by the poet and physician Justinius Kerner

in 1820, who reported on a case occurring in 1793 near Wildbad, and on a number of epidemics in various parts of Württemberg. The total number of cases involved was seventy-six, of which thirty-seven were fatal. (See Ostertag.[2]) In a second publication in 1822, the author reports ninety-eight additional cases with thirty-four deaths. Since that time a considerable number of poisonings have occurred in Württemberg after the eating of liver sausage, and a sausage known as "Schwartemagen," although in other countries, particularly in the northern part of Germany, such poisonings are rare. According to Ostertag the reason for this is primarily because of the enormous extent of the sausage industry and of sausage consumption in Württemberg (especially as regards these varieties), secondly because of the poor material used, and finally because of the primitive mode of manufacture in vogue at that time. The sausages were usually large in caliber, having been filled into pig stomach instead of into small gut. The smoking was therefore usually insufficient. The sausages also contained too much water. Since the methods of manufacture have been improved this form of meat poisoning has been of rare occurrence in Württemberg.

SYMPTOMS

The symptoms are very characteristic, and contrast sharply with those seen in the other forms of meat poisoning. Whereas, in the latter the symptoms are mainly gastrointestinal, in botulism they are almost

wholly referable to the central nervous system (v. Ermengem), and consist of secretory disturbances and symmetrical motor paralyses. The latter, either total or partial, affect especially the muscles supplied by the cranial nerves, hence one sees disturbances of accommodation, ptosis, double vision, dysphagia, dryness in the mouth and throat due to inhibition of salivary secretion, aphonia, obstinate constipation, and retention of urine. In addition there are disturbances in the heart action and in respiration. Fever is absent. Aside from this there are no motor or sensory paralyses, and consciousness is unimpaired. Altogether the symptoms remind one strongly of atropine poisoning. As a rule they appear in from twenty-four to thirty-six hours after the infected meal, though sometimes the onset has been within four hours, or again not until the fourth day. Death is not uncommon, and is due to asphyxia of bulbar origin. Many of the other cases take a long course, of weeks or months. Even when the case terminates favorably, disturbances of vision and muscular weakness persist for some time. According to some statistics collected by Senkpiehl, out of 412 cases which occurred from 1789 to 1886, there were 165 deaths, making a mortality of about 40%. Autopsy findings are almost entirely negative; usually all that can be seen is some hyperæmia of the organs.

Although no bacteriological examination was made, the following is probably an instance of this form of poisoning. It was reported by Sheppard,[97] in 1907,

and relates to three fatal cases. A party of three went on a camping trip and took with them as part of their rations two cans of pork and beans. About eighteen hours after eating of these beans, all three became ill, the symptoms in all being much the same. "There was," says Sheppard, "an entire absence of the usual gastrointestinal symptoms from first to last, no pain or sensory disturbance and no elevation of temperature. The first complaint was disturbance of vision, diplopia or a mistiness while looking in certain directions. Ptosis was present in two of the cases. Thickness of speech and difficulty in swallowing, which later became impossible, were present in all. Difficult breathing was also a constant and common symptom, and a general failure of muscular power; the whole picture being one of a gradually developing motor paralysis. A profuse secretion of mucus in the throat was a source of great distress, as owing to the paralyzed condition of the throat muscles it was impossible to get rid of it. The pulse until near the end was but little altered, except for a marked quickening on any movement of the body. Temperature was normal or subnormal. Free purgation could not be secured, owing presumably to the paralyzed condition of the intestines in common with the rest of the body. The kidneys secreted freely an apparently normal urine, but no examination of it was made. The mental condition was clear and undisturbed to the last, except for an unnatural irritability shown at times when the patients were making an effort to say something which

could not be understood." Death occurred on the fourth day.

Investigation showed that the beans had been purchased four months previously, and had been kept, for a time at least, in rather a warm place. This would give infecting bacteria opportunity to develop and produce abundant toxin. That such was the case was shown by feeding what was left of the beans to twelve chickens. Nine of these died, and the other three were made ill.

ETIOLOGY.

The cause of sausage poisonings was found by v. Ermengem[51] to be an anærobic bacterium which he termed B. botulinus, and which produces a highly poisonous specific toxin. This organism was discovered in a ham which had caused fifty cases of "sausage poisoning" in Ellezelles in 1895, three of the cases ending fatally. The bacillus was found in the intermuscular connective tissue in the form of spores, but was absent in the fat. The same organism were found in the spleen and gastric contents of the fatal cases, though here their number was much smaller. The ham was derived from a hog which appeared perfectly sound; and that this was the case was indicated by the fact that the rest of the pork (eaten fresh), as well as the other ham of the same animal, were eaten without producing any toxic symptoms whatever. Further investigation showed that the poisonous ham had been corned and had lain on the bottom of the cask covered with brine; the unaffected ham had been placed on top

of this, but had not been covered. In the latter, therefore, conditions for anærobic growth were not favorable. The poisonous ham was not putrid, but gave off a marked rancid odor not unlike that of rancid butter. It was also a little discolored, and somewhat softened. Watery extracts of the ham were injected into a number of animals and produced typical symptoms of botulism. In cats the symptoms consisted of marked mydriasis, disturbances of salivary secretion, various kinds of pareses, drooping of the tongue, aphonia, dysphagia, retention of urine, fæces, and bile. In pigeons there was paralysis of the wings, ptosis, unequally dilated pupils; in monkeys, guinea-pigs, rabbits, and mice there were symptoms of general or partial paralysis.

BACTERIOLOGY

Bacillus botulinus is quite a large organism, having rounded ends, and producing oval spores situated in the end of the bacillus. Motility is slight, and is accomplished by four to eight very fine peritrichous flagella. The bacillus stains according to Gram, *i.e.*, is Gram positive. It is a strict anærobe, and grows luxuriantly in glucose agar and bouillon with the development of gas. The cultures have a rancid smell strongly resembling the odor of butyric acid. The resistance of the spores is relatively slight; cultures containing spores are certainly killed by heating for one hour to 80° C. The bacillus does not develop in media containing over 5 to 6% salt; hence it should not develop in properly corned meat, where the brine

contains 10% salt. Bacillus botulinus produces a strong toxin. Filtered cultures injected into susceptible animals such as rabbits, guinea-pigs, mice, cats, and monkeys in doses even as small as 0.0001 cc., produce symptoms of paralysis. Larger doses 0.1 to 0.5 cc. are rapidly and intensely poisonous. After an incubation period of several hours, the animals are often seized with dyspnœa and convulsions, they fall on their sides paralyzed, and die as a result of rapid respiratory paralysis in from one-quarter to half an hour. The greater the dose of toxin, the more rapid and severe the development of the symptoms. There is, however, a minimum beyond which the period of incubation is not affected. No matter how large the dose, this is never less than from six to twelve hours. The symptoms have the character of a pure intoxication, without any multiplication of the bacilli in the body taking place. In contrast to what is seen in most other toxins, the toxin of this bacillus produces its symptoms not merely after subcutaneous or intravenous injections, but also and especially after feeding per os. In the animals dead after such poisoning, degeneration of the ganglion cells of the anterior horn, and of the bulbar centers (motor oculi) are observed. These are the organs which the clinical course of the disease would show were implicated.

Kempner [52] immunized animals with botulism toxin and succeeded in preparing an antitoxic serum which possessed protective and also some curative power in animal experiments.

Roemer [58] confirms all the above observations. In an investigation in 1900 he isolated B. botulinus from a ham which had given rise to typical symptoms of botulism poisoning in four persons. This ham came from a sound hog, and had been corned. So far as could be learned the ham lay on top, but was covered with brine. After five weeks it was noticed that bubbles of gas appeared in the brine. The fleshy part of the ham for the most part was normal in color; here and there, however, there were a few bluish-gray areas, softer in consistency and moist. The odor was not putrid, but rather sharply rancid, resembling the odor of butyric acid. No bacilli were found in the fat or in the healthy tissue, either by microscopical or cultural examination. From the discolored areas, however, B. botulinus was isolated, and also two ærobic bacteria. One of these was a large coccus, the other a bacillus belonging to the hay bacillus group.

Van Ermengem also found ærobic bacteria associated with the bacillus botulinus, and it is likely that it is this association which enables the latter organism to develop anærobically in the brine. Roemer found that the bacillus does not produce its poison in the living animal body, and that it does not multiply either at the site of injection or in the internal organs, or in the intestine. In other words, it is a saprophyte, and produces the symptoms entirely through the toxin which it produced in the infected meat. Man is poisoned because the toxin is absorbed from the gastrointestinal tract. Van Ermengem classes bacillus botu-

linus with the "pathogenic saprophytes," in order to show that although it cannot develop in the living animal body, it is still pathogenic through the poison which it produces in the infected food.

DIAGNOSIS.

The diagnosis of botulism can only be established bacteriologically through miscroscopical examinations of the suspected meat, through anærobic cultures on glucose agar plates or glucose gelatine, and through animal experiments. The latter will include feeding of the meat to mice, feeding and inoculating watery extracts into rabbits and guinea-pigs, and testing the toxicity of a several days' bouillon culture and of the filtrate from such a culture on these animals.

PROPHYLAXIS.

In discussing the prophylaxis of this variety of poisoning, v. Ermengem advised that such foods as are especially liable to botulinus infection, such as sausages, salt pork, preserved meats, etc., should never be eaten uncooked. In addition to this it is important that all decayed foods whose greasy appearance, rancid or butyric acid-like odor, consistency, or other abnormal constitution excites suspicion, should be destroyed. In corning meat the brine should always contain sufficient salt, at least 10%, for bacillus botulinus cannot develop in brine of this concentration. In the manufacture of sausages and bologna, only sound meat and organs should be used which have been thoroughly cooked. The gut used for sausage casing should be

carefully cleansed, if necessary, with the addition of a harmless antiseptic. Schilling [54] reports finding pieces of fecal masses, straw, and hairs in such sausage casing. In one meter of gut he found what was equal to 2 to 16 grams fluid fæces. It it well to avoid sausage casing of very large caliber (beef or hog stomach), for then it is difficult to thoroughly smoke the sausages. Attention should be paid to proper smoking. This should be carried out in suitably constructed chambers, and continuously until the sausages are sufficiently firm and dry. The sausages should not contain more than 30 to 35% water. In countries where sausages are much eaten good results have been achieved by issuing proper instructions to the public concerning the dangers from this form of poisoning, and formulating suitable laws to carry into effect the above recommendations.

TREATMENT

The treatment of botulism is essentially symptomatic, and consists in washing out the patient's stomach, administering stimulants subcutaneously, and employing artificial respiration if necessary. The bowels may be irrigated with hot saline infusions. If available, one may also employ an antitoxic serum. Such a serum possesses some curative value in animal experiments, and it can therefore be employed even after the onset of symptoms. Wassermann has recently begun to supply such a serum from the Institute for Infectious Diseases in Berlin.

IV

POISONING THROUGH FISH AND MOLLUSCS

FISH POISONING

THESE poisonings, too, must be divided into such in which the poison exists in the living animal and such in which the poison develops subsequently.

True poisonous fish are rare here, and are mostly observed in tropical countries. Poisoning occurs only when the fish is used for food. In many species, *e.g.*, in the Japanese fish "Fugu," the roe is poisonous, and when eaten by man give rise to choleraic symptoms, paralyses, and convulsions which are rapidly fatal. It is said that if the roe and all ovarian tissue is carefully removed from the fresh fish, no harm follows the eating of the fish itself. The poison is usually not destroyed by boiling. In Germany the roe of barbs (*Cyprinus barba*) is poisonous when eaten, especially in the month of May, but the symptoms usually terminate favorably. It is said that the roe of pike, and the meat of sturgeon (*acipenser huso* and *acipenser Ruthenus*), are also poisonous during the spawning season. According to Kobert [63] the liver, and especially the bile, of a number of fish is poisonous.

CLINICAL TYPES OF CASES

The greater number of fish poisonings, however, are due to bacterial infection or to intoxication following the eating of diseased fish or of fish whose meat has undergone post-mortem putrefaction. Ulrich[64] describes an outbreak of fish poisoning in Zürich in 1904 which was probably caused by some pike shipped from the coast. The transportation of the fish had taken some days; and they were kept from twenty-four to thirty-six hours and longer after cooking. The symptoms were gastroenteric and typhoidal in character, and varied in intensity according to the interval elapsing from the time of catching the fish until they were eaten. The longer the interval, the severer the symptoms. Furthermore, it was noticed that some sound fish kept on the same platter as the poisonous ones also become infected, and were fully as dangerous as the latter. Wyss and Silberschmidt were able to isolate the paratyphoid bacillus B. from the blood of two of the persons who died. The blood of the other patients agglutinated this bacillus. It was impossible to determine whether the living fish had been infected with the organism. It may be assumed, however, that the flesh of diseased fish is more suitable for the development of pathogenic bacteria than is that of healthy fish. Ulrich found that raw fish, especially in summer, contains a large number of bacteria, particularly members of the colon and proteus groups. Ordinary boiling does not suffice to kill all

the bacteria, and if the fish is kept for some time after cooking, especially in summer, bacterial multiplication is rapid. When large numbers of bacteria are thus taken with the food, severe gastric disturbances may be produced. In this respect colon infection is much more dangerous than proteus, for with the latter, putrefactive changes (putrid odor) soon manifest themselves, which is not the case in colon infections. The paratyphoid bacillus isolated from the fish grows luxuriantly in fish meat. Owing to the rapid multiplication of bacteria in cooked fish at warm temperatures, Ulrich regards the eating of fish in summer as dangerous when more than twenty-four hours have elapsed since the cooking.

In 1906 an outbreak of fish poisoning was reported by Abraham.[65] This embraced twenty-eight cases which developed eighteen hours after eating some pike. The symptoms were fever, intestinal colic, mild diarrhœa, and nausea. After two to three days the fever subsided, the colic and diarrhœa ceased, and the patients were entirely well at the end of a week. The piece of fish that was left over showed nothing abnormal either in color, odor, or taste. The bacteriological examination made by Neisser disclosed the presence of a bacillus belonging to the paratyphoid group, type Aertryk. This organism produces a toxin which withstands considerable heating. Serum examinations conducted on the blood of a number of the patients gave positive results with this bacillus. No such bacilli could be found in the stools of the pa-

tients. In this case it was believed that the fish had become infected during life, the bacilli lodging in the flesh, and thus causing the infection in the persons who ate of the meat. In view of the fact that the pike is a voracious feeder, spends considerable time about the mouth of sewers, and also feeds on other cadavers, it is quite conceivable that pathogenic bacteria such as the paratyphoid bacilli may gain entrance into his body.

In addition to these two forms of poisoning, one meets with cases of fish poisoning whose course is much like that described under botulism. Such cases usually follow the eating of canned fish or lobster when the can has remained standing open for some time. In some instances it is probable that the poison was contained in the mayonnaise dressing. It is not unlikely that the numerous attacks of indigestion following the eating of lobster salad are in part due to such poisons.

Severe illness has often followed the eating of crabs. It has been found that poisonous substances sometimes develop in boiled crabs, on standing, even before the appearance of a putrid odor. This is especially true when the animals have been boiled after death (Schneidemühl[4]). The symptoms which have been observed with this form of poisoning were lassitude, dragging pains in the back, and painful stiffness in the limbs.

Convalescence was protracted, sometimes lasting months.

MUSSEL POISONING

In 1885 an outbreak of poisoning occurred in Wilhelmshaven following the eating of common mussels (*Mytilus edulis*). There was no evidence of putrefaction. In most of the cases, the symptoms appeared in from one-quarter to one-half an hour after the meal, and consisted in a feeling of constriction in the throat, itching of the extremities, dizziness, marked weakness. In the fatal cases death occurred in from two to five hours. At the autopsy Virchow constantly found an enlarged spleen, and fatty degeneration of the kidney and liver. Fatal cases of this form of poisoning have been reported by other observers. Brieger succeeded in isolating a poisonous alkaloid which he termed mytilotoxin, and whose action was very similar to that of curare. He found that the poison could be destroyed by boiling with water containing three to five grams of sodium carbonate per liter. It is not known how this poison originates. In most instances it is said that the poisonous mussels came from stagnant water. Schmidtmann found that sound, non-poisonous mussels placed in the water of the canal from which the poisonous mussels had been derived, acquired toxic properties; and conversely, when the poisonous mussels were kept in the clean water of the bay, they rapidly lost all trace of poison. He therefore assumed that bacteria present in the canal water gave rise to the poison in the mussels. Schmidtmann states that poisonous mussels possess a sweetish, nauseating,

bouillon odor, while sound mussels have the odor of fresh sea water. Lustig and Zardo isolated two different varieties of bacteria from mussels, and found the cultures virulent for animals. They did not establish the identity of these organisms.

SNAIL POISONING.

A kind of marine snail, *Murex bradatus*, has also been the cause of poisonings of this kind. Such an outbreak was reported in 1900, in Isola, by Galeotti and Zardo.[66] Forty-three persons were affected, the symptoms being severe vomiting, hæmaturia, convulsions, paralyses, and diarrhœa. In some of the severest cases constipation was observed. Five of the persons died, and the autopsy revealed the presence of nuerous hæmorrhages scattered through the subcutaneous connective tissue, the muscles, serous membranes, and heart. There was also fatty degeneration of the liver, heart, and kidneys. From a number of the other snails captured in the same locality the observers isolated a bacillus related to the bacillus of hæmorrhagic septicæmia. This organism was highly virulent and was toxic even in feeding experiments. Animals killed in this way exhibited pathological changes very like those found in the human autopsies. Investigation showed that this bacillus was probably a common normal inhabitant of the molluscs in those waters, and that it in some way acquired pathogenic properties for humans.

It must not be forgotten, however, that many of

these mussel and sea snail intoxications are due to changes caused by proteus and other putrefactive bacteria.

POISONING THROUGH OYSTERS

Oysters have also frequently been the cause of intoxications, especially when the oyster beds are located near the mouths of sewers, or when the oysters are fattened in such waters. According to Bardet, practically all oysters are diseased during the summer months. Furthermore, oysters very readily spoil, and the eating of such oysters is very dangerous. The symptoms produced vary considerably. Sometimes the poisoning takes the form of an urticaria, sometimes that of a severe gastroenteritis, and fatal cases running a course like that of botulism have been described. The bacteriological investigations which have been made in these cases have not yielded definite results. Typhoid fever and cholera may also be spread by the eating of infected oysters, especially when the waters from which the oysters come are polluted by sewage. A number of outbreaks of typhoid fever, or a disease resembling this, have been traced to the eating of oysters, but in only very few instances was the typhoid bacillus really demonstrated. On the other hand, bacillus coli has repeatedly been found in oysters, and this has been taken as indicating fecal pollution. Other authors have interpreted this quite differently, saying that colon bacilli are a common normal inhabitant of these shell fish. Bacilli belonging to the proteus group have also been demonstrated in oysters, and Vivaldi

and Rodella[67] isolated a bacillus resembling B. coli, and belonging to the group of capsule bacilli. This was pathogenic for humans. It is absolutely necessary that oyster beds be located only where fecal contamination from sewers, etc., can be excluded. Furthermore, it is important that oysters, clams, and other shell fish be eaten only when fresh. Travelers should be particularly careful, especially in southern countries, for many a person has become infected with typhoid fever or suffered from some grave intestinal disorder by the indiscriminate eating of shell fish while traveling. One should always hesitate when the oysters are offered at especially low prices, for this is often merely a means of disposing of stale or otherwise undesirable wares. Attention should be paid to signs indicating dead oysters (gaping shell), and to indications of putrefaction (discoloration and softening, with a black ring on the inner side of the shell. Vagedes[68]). Poisoning through oysters is especially frequent during the summer months, whence comes the custom of eating them only during months whose names contain an R.

TREATMENT

The treatment of these poisonings does not differ in general from that outlined under meat poisoning on page 47, and under botulism on page 71. Cases running a typhoidal course should be treated exactly like typhoid fever, and the same precautions should be observed.

V

POISONING THROUGH CHEESE

CLINICAL TYPES OF CASES

POISONING due to the eating of infected cheese has been reported repeatedly. The ordinary symptoms are diarrhœa and vomiting, and in severe cases vomiting of blood, rectal tenesmus, collapse. In some instances there were disturbances of vision, or taste, dryness in the throat, obstinate constipation, etc., symptoms, in other words, much like those of botulism. As a rule nothing peculiar was noted about the taste of the cheese, except that it was sometimes somewhat bitter. The cause of this form of poisoning was formerly believed to be a poisonous alkaloid discovered by Vaughan, and termed by him tyrotoxicon. But recent investigations have repeatedly shown bacteria to be the cause of such poisonings. In an outbreak described by Vaughan and Perkins,[60] twelve persons became ill three to six hours after eating, with nausea, vomiting, pains in the abdomen, and threatened heart failure. Some of the patients had dilated pupils and some even were delirious. The authors were able to isolate a bacillus from the suspected samples, and found this to be pathogenic for the ordinary laboratory animals. The bacillus produced a strong toxin. By

mistake a patient was injected with ten drops of a sterilized culture of the bacillus in milk. Within thirty minutes the patient complained of dizziness, vomited freely, and had severe diarrhœa. Two hours after the injection he was almost entirely deaf, and delirious; and after another hour the patient became stuporous, with cold feet and hands, and imperceptible pulse. After injections of strychnine the patient gradually recovered, so that within twelve hours after the onset all threatening symptoms had passed. It was two days, however, before the patient was again able to move about the room. Curiously, guinea-pigs withstood much larger doses of this poison than ten drops. In fact, it required 1 to 2 cc. of the poison to produce toxic symptoms. The poison resisted heating to some extent. Fifteen minutes' heating to 100° C. did not entirely destroy its toxicity. The bacillus, on the other hand, was easily killed at much lower temperatures, and sterilization of the milk therefore inhibits further production of the poison.

According to Holst,[70] a cheese known as "Knetkäse," often produces attacks of acute gastroenteritis. The cause of this illness was found to be a bacillus related to the colon bacillus. This organism is very highly virulent for rabbits and calves, and seems to be similar to the bacillus of calf dysentery, described by Jensen. The cheese may become infected in a number of ways. The organism may reach the cheese through uncleanliness of the persons engaged in its manufacture, especially during the kneading process, or during

the handling in transportation, or the bacillus may have gotten into the milk by being derived from a cow suffering from some diarrhœal infection. Gaffky, for example, has shown that diarrhœal disease in man may be due to the drinking of milk from a cow suffering from diarrhœa.

Pflüger [71] described an outbreak of poisoning in which intense colicky pains, vomiting, diarrhœa with great prostration, etc., developed suddenly twelve hours after the persons had eaten some sour cheese. In some of the cases there were also disturbances of vision, diplopia, dryness of the mouth, dysphagia, and other symptoms resembling those of botulism. It is possible that anærobic bacteria similar to the B. botulinus are concerned in this form of cheese poisoning, but so far no investigations in this direction have been reported.

OTHER BACTERIA ASSOCIATED WITH CHEESE POISONING.

In an outbreak of suspected cheese poisoning, Peppler [72] isolated organisms identical with the bacilli of swine erysipelas. According to recent investigations, these bacilli can give rise to serious intestinal disturbances in man. Furthermore, tubercle bacilli have repeatedly been found in cheese, though their significance in the etiology of human infections is still open to question. According to Heim,[73] other pathogenic bacteria, such as cholera or typhoid, which may chance to get into cheese, usually die off within a few days.

In order to emphasize the importance of *bacterio-*

logical examinations in all these poisonings, the following account of a careful but disappointing chemical examination may not be amiss.[74] A piece of cheese to which decided toxic symptoms were correctly ascribed by Dr. Lartigau, was sent to Professor Gies for examination. The symptoms were similar to those given by Vaughan and Novy for poisonous cheese. A portion of the cheese was therefore extracted and examined for tyrotoxicon by Vaughan's method, with negative results. A portion of the extract was examined for proteins. The greater portion of the soluble protein in the extract was found to consist of deuteroproteose, with some peptone. After precipitating the proteose with alcohol, the filtrate yielded on evaporation relatively large amounts of leucin and tyrosin. It also contained some tryptophan. The unused residues, solid and liquid, were combined and examined by the Stas-Otto method for alkaloidal substances, with negative results. The authors conclude that "in all probability the poisonous matter in this particular case consisted of toxic proteose, although this was not suspected until practically all of the material had been used up. The seeming certainty that ptomaines were responsible for the symptoms noted had been entirely misleading."

TREATMENT

The treatment of these poisonings is entirely symptomatic, and depends on the clinical type of case. (See pages 47 and 71.)

VI

POISONING THROUGH ICE CREAM AND PUDDINGS

HISTORICAL

Poisoning due to the eating of vanilla sauce or vanilla ice cream is remarkably frequent. The symptoms usually come on within one and one-half or two hours, and consist in severe vomiting, pains in the stomach and abdomen, diarrhœa, and signs of collapse. At one time pharmacologists ascribed the poisonous action to the vanilla used for flavoring, but during an outbreak of this form of poisoning in 1894, M. Wassermann [75] was able to demonstrate that a bacterial intoxication was involved. The poisonous dish contained milk, eggs, sugar, and vanilla-sugar (10 grams sugar containing 20% vanilla). The dish was prepared in the evening, and was then kept, uncovered and at room temperature, in the pantry until the following noon. Neither the vanilla nor the vanillin produced any symptoms when tested in animals. It was found, however, that vanillin, the active principle in the vanilla sugar, through its reducing action, favors the growth of anærobic bacteria. We know that milk often contains bacteria which require anærobic conditions, are pathogenic for man, and withstand considerable heat-

ing. Under these circumstances we can understand that in the preparation of a pudding such as that described, we have conditions altogether favorable for the development of these bacteria and the production of their poisons.

Vaughan,[76] in studying an outbreak of poisoning due to the eating of vanilla ice cream, also proved that the vanilla employed was harmless. The milk used for making the ice cream was fresh, and some lemon ice cream made from the same lot of milk was absolutely harmless. The custard mixture used for both kinds of ice cream was prepared in one lot, and then divided into two portions. One was flavored with vanilla, the other with lemon. According to the investigations of Wassermann, the different behavior of the two kinds of cream is to be explained by the fact that vanillin favors the growth of anærobic bacteria, while lemon extract even acts as an antiseptic.

In studying an outbreak of poisoning in 1895, following the eating of ice cream, Vaughan isolated the same colon-like bacillus already mentioned in the outbreak of cheese poisoning.

BACTERIOLOGY

In recent years paratyphoid bacilli have been demonstrated as the cause of poisoning through farinaceous puddings. As already stated, Fischer[25] pointed out the rôle played by milk in spreading paratyphoid infection. In 1904, Vagedes[77] observed an outbreak of poisoning in Berlin, which he traced to the eating of

farina pudding. There were seven cases in all, and the symptoms, which came on within a few hours after eating the pudding, consisted of intestinal catarrh accompanied by fever and copious diarrhœas. One of the cases ended fatally, and showed merely a distinct swelling of Peyer's patches. The bacteriological examination of the blood-tinged stools and mucus showed the presence of paratyphoid bacilli type B. The same organism was isolated from the spleen, liver, and kidney of the fatal case. In cultures this organism produced a strong poison which was quite resistant to heat. The serum of the patients agglutinated this bacillus; in fact, seven months after the attack the serum of one of the patients still showed agglutinating power. Typhoid bacilli were also agglutinated by the serum of these cases, but only in much more concentrated dilutions. It was impossible to find these paratyphoid bacilli in the food because nothing was left to examine. Nevertheless all the clinical, epidemiological, and bacteriological data pointed strongly to the farina pudding as the cause of the poisoning. The pudding was prepared with farina, zwieback, apples, milk, sugar, vanilla powder, and three duck eggs. How the bacilli got into the pudding could not be ascertained. It was shown that neither the milk, vanilla powder, nor the zwieback were infected. Perhaps the duck eggs were to blame, for an examination of a number of other duck eggs shows that some of them contained a considerable number of bacteria. It is probable that decomposed eggs may also lead to poisoning,

for eggs constitute an admirable medium for the growth of bacteria, and of course for typhoid and paratyphoid bacilli. Moreover, Lange[78] has pointed out that bacteria are able to penetrate the intact shell and so reach the egg yolk. Naturally this is still more liable to occur when the shell is cracked. It is well to bear in mind that eggs may be the carriers of infection in these cream and pudding poisonings.

An outbreak of poisoning is reported by Curschmann[27] in which twenty-two persons were taken ill after eating a pudding composed of milk, eggs, sugar, gelatine and vanilla, and served with raspberry sauce. The milk had previously been boiled; the pudding had been prepared the evening preceding the meal, and had been kept in a cool place. When eaten there was nothing whatever about taste or odor to excite suspicion. All those who ate of the pudding were attacked in five to six hours with severe abdominal pains, vomiting, and diarrhœa; usually there was also high fever and rapid pulse. Some of the patients were drowsy. There was no enlargement of the spleen. One of the cases ended fatally in collapse, and came to autopsy. Marked inflammatory changes were found in the stomach, and to some extent also in the intestine. There was also some change in the kidney parenchyma.

From the remains of the pudding, and from the stools of several of the patients, as well as from the liver of the fatal case, Curschmann was able to isolate a bacillus belonging to the enteritidis group; this organism was virulent for mice. It was impossible to

learn how the bacillus had gotten into the pudding, or which ingredient had carried the infection. The vanilla was free from such bacteria; there was no more of the milk left to examine; Curschmann believed that in some unknown way the pudding had become infected between the time of making and of eating. Since we know practically nothing concerning the occurrence of enteritidis bacilli in nature outside of the animal body, it is impossible to say anything more concerning the spread of such infections.

In 1905 Levy and Fornet [79] studied an outbreak of illness affecting seven persons in Strassburg. The patients were attacked with vomiting and severe diarrhœa, in some there were also a typhoid roseola and enlargement of the spleen. The authors were able to isolate paratyphoid bacilli, type B., from the stools of all the patients. The symptoms appeared almost simultaneously in all the members of the family, and there is no doubt that the infection was due to food. Investigation showed that only two articles of food could be implicated, namely, some liver sausage and some vanilla farina pudding. Examination of the liver sausage failed to show any paratyphoid bacilli, and there was nothing left of the pudding to examine. Examination of the farina itself and of the vanilla beans yielded negative results.

It thus appears that the paratyphoid bacillus B. plays an important part not only in meat poisonings, but also in other food poisonings. Thus far, however, we have absolutely no information concerning the man-

ner in which the bacilli get into the foods. It is suggestive that almost always the suspected foods have contained milk and vanilla. We may recall that Fischer [41] in studying an outbreak of paratyphoid infection in Futterkamp found that this was due to the drinking of milk from cows suffering from gastroenteritis. He also isolated the paratyphoid bacillus from the milk of these animals. In the paratyphoid infections associated with the eating of farina puddings, we may therefore assume that the milk was probably the carrier of the infection. E. Klein [80] inoculated thirty-nine specimens of milk into guinea-pigs, using the sediment from 300 cc. of milk. In ten of the specimens the inoculation produced little foci of pus in the spleen of the animals, and further examination revealed the presence of bacillus enteritidis in these foci. Feeding a milk culture of these bacteria to guinea-pigs killed half of the animals in five days. As far as could be ascertained none of the cows yielding this milk were ill.

We have already called attention to the occurrence of bacilli of the paratyphoid group in the intestines of normal animals. It is obvious that such bacilli, when they are discharged in the fæces, especially about a dairy barn, can quite readily find access to the milk. (See also Cheese Poisoning, page 85.)

PROPHYLAXIS

So far as the prophylaxis against this form of food poisoning is concerned, very few positive statements

can be made. Certainly all such puddings, etc., should be thoroughly boiled, and, after rapid cooling, should be eaten soon after cooking. If they are to be kept over for some time, they should be carefully covered, and then placed in a cool place, preferably on ice.

TREATMENT

(See remarks on page 83.)

VII

POTATO POISONING

HISTORICAL

LARGE outbreaks of poisoning through potatoes and potato salad seem to be of frequent occurrence among troops. Schmiedeberg [81] reports on an outbreak which occurred in 1892, in which 357 members of one battalion were attacked with frontal headache, severe colicky pains in the stomach and abdomen, vomiting, diarrhœa, prostration, and slight delirium. In a few of the cases the symptoms were quite threatening, cyanosis of the lips, markedly dilated pupils, syncope, rapid pulse, etc. In the severe cases there was some rise in temperature. Investigation pointed to new potatoes as the cause of the outbreak.

About the same time a similar outbreak occurred in a battalion belonging to an entirely different garrison. Ninety men were suddenly attacked with frontal headache, abdominal pains, diarrhœa, prostration, and dizziness. Some of the patients had a little rise in temperature, but none had any dilatation of the pupils or increased frequency of the pulse. The probable cause of the outbreak was believed to be potatoes. Although somewhat soft and watery, the potatoes were generally quite ripe.

In 1893 a third outbreak of this form of poisoning was reported from still another garrison, in which 125 men were affected with symptoms like those just enumerated. All the persons made a good recovery.

Schmiedeberg also cites an outbreak described by Cortial, occurring in Lyons, in July, 1888. This likewise occurred among the soldiers and affected 101 individuals. The main symptoms were prostration, colicky pains, diarrhœa, fever, and headache. Some of the cases had dilated pupils. The outbreak was ascribed to the eating of potatoes, both new and old sprouting ones. After the delivery of new potatoes was stopped, no more cases occurred. A dog that had eaten three times of these potatoes suffered from diarrhœa for a week. None of the cases in this outbreak ended fatally.

In 1898, E. Pfuhl [82] reported an outbreak of poisoning among some soldiers. There were fifty-six cases, and in almost all of them the symptoms began a few hours after dinner. Most of the patients had chills, fever, headache, severe abdominal pains, diarrhœa, nausea, drowsiness, lassitude, and some complained of a scratchy feeling in the throat. The pupils were not dilated, and the fever lasted only for about three days, dropping by crisis. The outbreak was ascribed to the eating of salted potatoes.

SOLANIN POISONING

At one time all these cases were regarded as instances of solanin poisoning. Schmiedeberg, however,

pointed out that this could be accepted only when the potatoes could be shown to contain sufficient solanin to produce symptoms of intoxication. According to the investigations of Meyer [83] the solanin content of potatoes in December and January is 0.04 g. per kilo of unpeeled potatoes, in March and April it rises to 0.08 to 0.096 g., and in May, June and July to 0.100 to 0.116 g. The amount of solanin, however, which is necessary to produce poisoning is stated by Clarus to be 0.2 to 0.4 g., so that the amount contained even in a whole kilogram of potatoes is insufficient to produce the symptoms described.

This question has been carefully studied by Wintgen.[84] In his extensive investigations he found large fluctuations in the solanin content of sound potatoes (0.017 to 0.08, g. per kilo), but the quantity was always small. When the potatoes were stored for some time, even when they sprouted, no increase in solanin was observed provided the sprouts were carefully removed. On the other hand, in diseased potatoes and such as are vigorously sprouting, the solanin content may be considerably higher. Thus Meyer found 0.58 g. per kilo, in the little dwarf potatoes which had developed from the sprouts of old potatoes, and in old potatoes which were much shrunken and discolored near the margins, he found as high as 1.34 g. solanin per kilo. Wintgen was unable to discover any marked difference in the solanin content of sound and diseased potatoes. The distribution of solanin in the potatoes is not at all regular; in the peel the amount is very

high, and toward the center of the potato the amount constantly decreases. Weil [85] stated that the quantity of solanin in the potato increases under the action of bacteria, and claimed to have isolated from diseased potatoes, two species of bacteria which possessed this power. Wintgen, however, was unable to confirm this statement.

Poisoning with potatoes rich in solanin differs somewhat from poisoning with pure solanin. In the former there are acute gastrointestinal symptoms and fever, in addition to the symptoms observed in pure solanin poisoning. The reason for the occurrence of these symptoms in the former condition is because the swollen starch hinders the rapid absorption of the poison, giving the alkaloid time to reach the lower part of the intestinal tract, where it causes more or less marked diarrhœa in addition to the vomiting.

In the outbreak described by Pfuhl,[82] the peeled, raw potatoes contained 0.38 g. solanin per kilo, and the peeled boiled ones, 0.24 g. The soldiers eating a large portion (1 kilo) of potatoes thus took up 0.30 g. solanin, a quantity which is sufficient to produce considerable toxic disturbances. These quantities of solanin, however, are very unusual, and one is not justified in charging an outbreak of potato poisoning to the solanin unless careful examination shows that the solanin content was actually very high. As a matter of fact, these examinations have hardly ever been carried out.

BACTERIA ASSOCIATED WITH POTATO POISONING

It is extremely probable that bacterial decomposition of potatoes by proteus bacilli, an observation first made by Dieudonné,[86] is a much more frequent cause of potato poisoning. During an encampment at Hammelburg, in August, 1903, 150 to 180 soldiers suddenly became ill two hours after dinner with severe and repeated vomiting, headache, intense diarrhœa, more or less marked symptoms of collapse, slight cramps in the legs, etc. There was no fever. The symptoms began to subside after seven hours, except in a few cases, where there was slight delirium, symptoms of collapse, and convulsions. All of the cases, however, recovered. The outbreak was ascribed to potato salad, and this was accordingly examined bacteriologically, disclosing the presence of numerous proteus bacilli. Mice fed with the salad died in twenty-four hours with severe gastrointestinal symptoms; when examined at autopsy, only a few bacilli were found in the organs. Bouillon cultures of the bacilli thus isolated were not virulent for test animals; but when sterile potatoes were inoculated with the organisms, and grown for twenty-four hours at 37° C., a highly poisonous culture was obtained. Mice fed with this culture died in twenty-four to forty-eight hours. If the potato cultures were grown at 10 to 12° C., the resulting culture was unable to kill mice. This shows that this proteus bacillus produced poisons on the potato, but only at high temperatures. Furthermore, the bacillus was not directly in-

fectious or toxic, but only indirectly through the toxic substances produced from the potato.

It was impossible to learn how the bacilli got into the potatoes, though it was perhaps from the hands of the persons who did the peeling. The potatoes used for the salad were new and tender, but they had been boiled the night preceding the meal, then peeled, and kept until the next day in two large baskets in a room adjoining the camp kitchen. The weather during the night and on the next morning was sultry, a fact which favored the growth and development of the germs. The potatoes used in making the salad were examined for solanin content, but this was found to be low, 0.021 g. per kilo. The potatoes did, however, contain considerable water, and this favored their decomposition.

It is interesting to know that such poisonings have been rather frequent when the potatoes have been cooked and peeled on one day and then kept in large containers until made into salad on the next. In the summer this poisonous decomposition can take place with great rapidity, as is shown by the following interesting outbreak. In a certain battalion of troops the members of one company became ill after eating potato salad which had been standing for two hours, while several other companies, two hours previously, had eaten from the same lot of salad without any injurious effect whatever. These two hours, therefore, sufficed for the formation of the poisonous products.

It is very probable that many of the potato poison-

ings formerly described were not due to solanin, but to bacterial decomposition. As has already been pointed out, solanin poisoning can only then be assumed to have occurred when large quantities of this alkaloid can be demonstrated in the potatoes. Besides proteus other bacterial infections are probably carried by potatoes. Both typhoid and paratyphoid bacilli are known to grow very well on potato. These bacilli may get into the potatoes on the farm, or the potatoes may subsequently be infected by the infected hands of a bacillus carrier. In studying an outbreak of potato poisoning, therefore, the bacteriological examination should include a careful search for typhoid and paratyphoid bacilli. The technique of this examination has already been outlined on page 38.

PROPHYLAXIS

Prophylaxis against this form of poisoning consists in using the potatoes as soon as possible after cooking, and not to keep them over from one day to the next, especially in summer. Almost all the outbreaks of potato poisoning reported have occurred in July and August, and usually after the use of new potatoes. Owing to their larger content of water, these potatoes decompose much more readily than old ones. In order to guard against solanin poisoning, it is wise to cut out all sprouts, and to carefully peel the potatoes. It is important that the kitchen help in all large institutions (barracks, hospitals, hotels, etc.) be frequently examined by physicians in order to exclude bacillus car-

riers from this work. Furthermore, it is essential to insist on absolute cleanliness on the part of all who have the handling of the food. Since the typhoid and paratyphoid bacilli thrive readily on potato, infection with these germs, in the way just indicated, may not be at all uncommon.

TREATMENT

Treatment of potato poisoning is symptomatic, and consists in administering cathartics and stimulants, and in washing out the stomach. (See page 47.)

VIII

POISONING THROUGH CANNED GOODS

CANNING AND THE DESTRUCTION OF BACTERIA.

As the use of canned goods becomes more and more extensive, poisonings of this kind are increasingly frequent. This is especially true of canned meat, fish, and vegetables. In the process of canning, the can with its contents is heated in an autoclave for one-half to one hour at 112° to 120° C. The cans are made of thin sheet iron coated with tin. The seams are pressed, and soldered with a thin coating of solder. The cans are filled with the cooked meat or vegetables, the cover is put on and then the whole is sterilized in the autoclave. It is true that practically all bacteria are killed by the temperature employed, but sometimes the apparatus does not work properly, or there is some other slip, and then bacteria subsequently develop. With this, of course, comes the development of putrefactive products. When this is accompanied by the formation of gas, the top of the can presents a convex appearance (technically called a "blown" can), a sign of warning to the consumer. On opening such a can a foul-smelling gas escapes.

CANNED MEATS: CLINICAL TYPES OF CASES

Poisonings due to canned meats have been reported quite frequently. Thus Bochereau [88] reports a number

of such instances occurring among the troops of the French army. The nature of these is rendered very probable by the statement that of 21,151 cans examined, more than fifty were more or less "blown" and decomposed. Canned fish is also frequently the cause of poisoning, and this seems to occur especially with canned salmon, particularly when the fish is not eaten directly after the can is opened. We have already pointed that fish constitute a splendid medium for the development of bacteria, and so may easily become the carriers of bacterial poisons. The symptoms observed with poisoning by canned meat or fish are the same as have already been described under meat poisoning, and are either gastrointestinal or nervous (botulism) in character.

An instance in which canned pork and beans produced three fatal cases of poisoning of the botulism type has been reported by Sheppard.[97] (See page 64.)

CANNED VEGETABLES.

In recent years a number of outbreaks of poisoning have been reported following the eating of canned vegetables. In Darmstadt, in 1904, such an instance occurred in a cooking school, twenty-one persons becoming ill after eating bean salad. Eleven of these died. According to A. Fischer[89] the symptoms appeared twenty-four to forty-eight hours after the meal, and showed the typical characteristics of botulism. (See page 64.) Thus there were disturbances of vision, ptosis but no mydriasis, dysphagia, various motor

paralyses, mostly bilateral, increased frequency of pulse, etc. On the other hand, gastrointestinal disturbances, fever, sensory and mental disturbances were entirely absent. Death occurred with symptoms denoting bulbar paralysis, in two to fourteen days after the poisoning. The autopsy disclosed the usual signs of asphyxia, and hyperæmia and submucous hæmorrhages of the lower part of the intestines. Otherwise there was nothing peculiar. The cases which recovered had a very slow convalescence. The beans used for the salad had been canned by one of the cooks of the school, who herself was one of those poisoned. On opening the can a peculiar rancid odor, somewhat resembling that of Parmesan cheese, had been noticed, but as there were no signs of decomposition, nothing more was thought of this. The beans were very tender, "soft as butter," and were therefore not further cooked, the salad being prepared after merely rinsing the beans under the faucet. It was remarked that the rancid odor increased after the salad had stood a while. Only those who ate of the salad were poisoned.

In studying the cause of this outbreak, Landmann [90] examined some of the salad left over, and extracted this with physiological salt solution. After passing the extract through a Berkfeld filter the sterile filtrate was found to be highly toxic, 0.5 cc. injected subcutaneously into mice killing the animals with symptoms of general paralysis in twenty-four hours. Boiling the extract destroyed the poison. This harmonizes well with the fact that several persons who ate some

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of the salad which, through an oversight, had been placed on the stove and allowed to boil, were not poisoned. So also in another case, where some of the salad was eaten after moderate heating; the toxic symptoms developed very slowly, with, however, a fatal ending. Bacteriological examination of the salad revealed the presence of an anærobic bacillus which was identical with bacillus botulinus. This organism produced a strong poison in cultures, especially when grown at 24° C., so that white mice were killed with 0.000003 cc. and guinea-pigs with 0.0003. When grown at 37° C., on the other hand, the fatal dose for mice was 0.01 cc., and for guinea-pigs 0.1 cc. Gaffky also isolated the bacillus botulinus from this salad.

It was impossible to learn how the string beans had become infected. Landmann believed that the bacilli had been carried into the can along with some little piece of left-over meat such as might readily be found in any kitchen. He bases this view on the fact that thus far the bacillus botulinus has been found only in meats or dishes prepared from meat. It is also possible, however, that the spores of this bacillus were carried in on the beans from the field. In the canning as carried out in the cooking school the sterilization appears to have been incomplete; it is probable that in a regular canning factory where sterilization is carried out at 112° C., all of the spores would have been killed. It follows from what has been said that canned vegetables having the least rancid odor should be destroyed, and that even those appearing normal in every

way should always be boiled before serving. If this had been done in the case just described, the catastrophe would have been averted.

An extensive outbreak of poisoning due to the eating of string beans occurred in Leipzig in 1906, among the employees of a large department store. About 250 persons were affected, a few hours after the meal, the chief symptoms being abdominal pains, chills, nausea, headache, and dizziness. In some of the patients the symptoms were at once accompanied by diarrhœa; in others the diarrhœa came on during the evening or the following morning. The symptoms lasted two to four days, all the cases making a good recovery. The string beans were delicious, and showed nothing abnormal in taste or odor. They had been packed in several cans, which had been opened just before the meal, and then placed for a time in hot water (about 80° C.). Boiling was purposely omitted because the beans were so tender that they would have made a soft mush. The bacteriological examination made by Rolly[91] disclosed the presence of two species of bacteria, bacillus coli, and bacillus paratyphi, type B, both in large numbers. No anærobic organisms were isolated. The paratyphoid bacilli were pathogenic for mice and guinea-pigs when inoculated subcutaneously; in cultures the bacilli produced a toxin which was resistant to heat. The poisoning in his case was due to the toxin and not to the bacteria themselves, for otherwise the symptoms would not have appeared so suddenly, but would have required a period of incubation. Furthermore, in none

of the cases could paratyphoid bacilli be found in the stools.

CLINICAL TYPES OF CASES.

We see, then, that in poisoning due to canned vegetables the same two types of bacteria occur as are found in outbreaks of meat poisoning, namely, bacillus botulinus, which produces a poison readily decomposed by heat, and bacillus paratyphi, B, whose poison withstands heating. The first variety of poisoning can be guarded against by heating the food to boiling just before serving, although it is better not to eat any food about whose condition there is the least question. The second variety of poisoning cannot be safeguarded against even by prolonged boiling.

Belser [92] made careful bacteriological examinations of canned vegetables which had undergone decomposition, as was shown by the ballooned tops of the cans. He found that the change was due to a number of bacteria which were more or less resistant to heat, such as bacillus acidi lactici, and bacillus amylobacter. In almost every case the decomposed vegetables showed an increased acidity, though sometimes this was but slight. The bacilli were not virulent for mice. Bacillus proteus was also encountered; this organism grows especially well in the broth of peas and beans, and produces a toxin highly virulent for mice. Bacteria ordinarily occurring in water and soil have also been found in canned vegetables; among these are bacillus mesentericus vulgatus, and bacillus megatherium.

BACTERIOLOGICAL EXAMINATION

It is impossible to determine the absolute sterility of canned goods without a careful bacteriological examination. According to E. Pfuhl,[87] canned goods often contain bacteria without showing any ballooning of the top. The reason for this is that the bacteria infecting the can do not all produce gas. Furthermore, the appearance of the contents on opening the can may also give no hint of the presence of bacteria. Frequently all that is noticed is that the contents have a slightly sour odor, or one abnormally pungent. Where large purchases are made, it is therefore necessary always to have several cans, selected at random, examined bacteriologically. The goods should not be accepted if this examination discloses the presence of any bacteria. Pfuhl gives the following method to be followed in making the examination: The can is kept unopened in the incubator for eight to fourteen days. This gives the obligate and faculative anærobic bacteria opportunity to develop. Then the lid is carefully sterilized by flaming with alcohol, and a hole made with a sterile ice pick or other sharp instrument. Through this hole some of the meat juice and liquefied gelatine is sucked up with a sterile pipette and planted into different media both for ærobic and anærobic growth. The hole is then covered with a bit of sterile cotton, and the can returned to the incubator for another two or three days. This gives the ærobic bacteria time to develop. Then another set of cultures is made. The

second test may be omitted if bacteria develop with the first test.

PROPHYLAXIS

Schottelius [93] has repeatedly warned against the ever-increasing use of canned foods in the household, and we believe the warning fully justified. There is a distinct field of usefulness for this form of food, for example, in the feeding of armies in war, or in expeditions of various kinds. On the other hand, in temperate climates, as for example in Germany or in the United States, there is really no reason, aside from convenience, why canned foods should be used in the household. It seems not to be appreciated that canned goods are always inferior to fresh vegetables, fruits, etc.* Furthermore, just the fact that certain fruits or vegetables are out of season for part of the time each year, makes our appetite and appreciation of them all the keener when the season returns. If we had strawberries and cream every day of the year, we should soon be disgusted by the mere sight of them. Another disquieting fact is that at the present time we have no way of determining the age of the goods we are buying. It will be recalled that the proposal to date all canned goods met with determined opposition on the

*While the superiority of fresh foods is unquestioned, there is probably very little danger of food poisoning from fruits or vegetables properly canned by a reputable firm. In fact, it is likely that the "canning" as done in such an establishment (in steam sterilizers under pressure), is more efficacious than that done at home.

part of the large packers. Yet there is no doubt whatever that the danger of decomposition increases with age, and there is also a loss of flavor. Under no circumstances should the contents of a "blown" can be eaten. It is said that unscrupulous packers have taken "blown" cans which had been returned to them, punched a small hole in the cover to let out the gas, reheated the whole in the autoclave, resoldered the punch hole, and then resold such goods at a reduced price. It will be well, therefore, to refuse canned goods showing more than one soldered opening.

TREATMENT

(See pages 47 and 71.)

IX

METALLIC POISONS

HISTORICAL

THERE is still a widespread belief among the laity that epidemic outbreaks of food poisoning are often due to metallic poisons. As a matter of fact, this is rarely the case. Nevertheless, notable outbreaks of this nature have been known even in recent years. Thus the famous "beer epidemic" of Manchester and other English cities, in 1900, was due to the accidental presence of arsenic. At one time, too, the chief cause of poisoning through canned foods was thought to be metallic poisons, especially lead and tin, but K. B. Lehmann [94] declares that this is not true at the present time. In Germany the law prescribes that the tin used for tinning the cans shall not contain over 1% lead, and the solder not over 10%. Lead poisoning from this source is unknown in that country since the enactment of this law. Poisoning from tin is probably also very infrequent. According to Lehmann acute digestive disturbances may be caused by foods which contain large amounts of tin (100 to several hundred milligrams), but the symptoms are usually not severe. Freshly canned goods contain but little tin; the amount, however, gradually increases in time, so that

canned vegetables were found with from fifty or sixty mg. to 200 mg. per kilo. Canned meats contained all the way from fifty to 325 mg. The largest amounts were found in decomposed canned foods, for in these the acid which was formed in the decomposition dissolved the tin. Large amounts were also found in acid preserves, such as pickled herring. In one such instance 156 mg. of tin were found in 150 grams of the pickled fish. It is important, therefore, to insist that preserves containing considerable quantities of acetic, tartaric, or malic acid be packed only in glass or porcelain, and not in tin.

According to Lehmann the ordinary vegetable or meat preserves, which are not highly acid, do not appear to give rise to any metallic poisoning. What poisoning does occur is almost always of bacterial origin.

Copper poisoning was also formerly believed to play an important rôle in food poisoning. Lehmann[95] states that about 200 mg. copper must be swallowed before symptoms are produced in man, and 1200 mg. are needed to produce really threatening symptoms. It is difficult to see how such quantities of copper could be present in the food under ordinary conditions, and there is little doubt that most of the poisonings ascribed to verdigris and copper are really due to bacterial decomposition of the food. In fact, the symptoms reported in these cases are very much like those of the latter condition.

There is reason to believe that under certain condi-

tions, lead poisoning may occur quite readily. Thus earthen ware is sometimes coated with a cheap glaze containing large quantities of lead. On boiling fluids containing vinegar in such pots, considerable amounts of lead pass into solution. Thus, in some tests made in this direction, 100 to 700 mg. lead were extracted with the first boiling. Lehmann[96] believes that attention should be directed to this pottery, because the lead is given off for a long time. Perhaps these lead glazes are responsible for some hitherto unexplained cases of lead poisoning. Lehmann cites a case of lead poisoning observed by Halenke. Two women had cooked cranberries in an earthen pot, and had then made a cranberry tart. Soon after eating the tart both became ill, one very severely. Investigation showed that the glaze had been completely dissolved from the inside of the pot, and that a piece of tart contained about 160 mg. lead. Each woman had therefore consumed about 400 to 600 mg. lead in the form of lead malate. Approximately 1000 mg. lead had been given off by the glaze in the one boiling. It will be well, when investigating chronic lead poisonings whose origin cannot be traced, to think of cheap pottery with a lead glaze. The determination of the lead content can easily be undertaken by any physician by merely boiling the pot with 4% acetic acid for half an hour, and then passing sulphureted hydrogen through the fluid. A black discoloration or a black precipitate denotes the presence of lead.

It is well to remember, however, that metallic poi-

sonings through cooking utensils, etc., are quite rare, and one is justified in ascribing poisoning to this cause only if a chemical examination has really demonstrated large quantities of such a poison. The mere fact that the food was prepared, for example, in a damaged copper vessel, by no means justifies the diagnosis of copper poisoning. In fact, one should first think of bacterial poisoning, for this is much more likely to have occurred.

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